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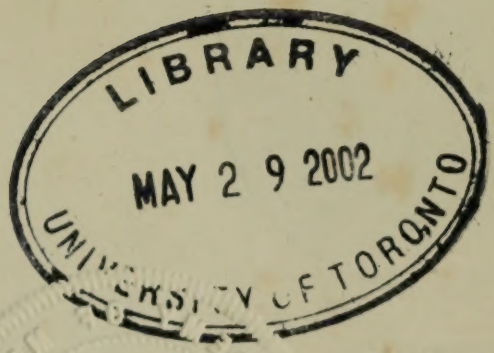

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T. PICKERING PICK
HENRY POWER
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1808. MATTHEW BAILLIE, M.D.
1810. SIR HENRY HALFORD, Bart., M.D., G.C.H.
1813. SIR GILBERT BLANE, Bart., M.D.
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1823. JOHN ABERNETHY
1825. GEORGE BIRKBECK, M.D.
1827. BENJAMIN TRAVERS
1829. PETER MARK ROGET, M.D.
1831. SIR WILLIAM LAWRENCE, Bart.
1833. JOHN ELLIOTSON, M.D. (First President of the Royal Medical and Chirurgical Society, 1834)
1835. HENRY EARLE
1837. RICHARD BRIGHT, M.D., D.C.L.
1839. SIR BENJAMIN COLLINS BRODIE, Bart., D.C.L.
1841. ROBERT WILLIAMS, M.D.
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1849. THOMAS ADDISON, M.D.
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1861. BENJAMIN GUY BABINGTON, M.D.
1863. RICHARD PARTRIDGE
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1867. SAMUEL SOLLY
1869. SIR GEORGE BURROWS, Bart., M.D., D.C.L.
1871. THOMAS BLIZARD CURLING
1873. CHARLES JAMES BLASIUS WILLIAMS, M.D.
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1881. ANDREW WHYTE BARCLAY, M.D.
1882. JOHN MARSHALL
1884. GEORGE JOHNSON, M.D.
1886. GEORGE DAVID POLLOCK
1888. SIR EDWARD HENRY SIEVEKING, M.D., LL.D.
1890. TIMOTHY HOLMES

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1887 Flower, William Henry, C.B., LL.D., F.R.S., Director of the Natural History Department, British Museum, Cromwell road.

1887 Foster, Michael, LL.D., F.R.S., Professor of Physiology in the University of Cambridge.

1883 Frankland, Edward, M.D., D.C.L., Ph.D., F.R.S., Corresponding Member of the French Institute; The Yews, Reigate Hill, Reigate.

1868 Hooker, Sir Joseph Dalton, C.B., M.D., K.C.S.I., D.C.L., LL.D., F.R.S., Corresponding Member of the Academy of Sciences of the Institute of France; The Camp, Sunningdale.

1868 Huxley, Thomas Henry, D.C.L., LL.D., F.R.S., Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.

1878 Lubbock, Sir John, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Hayes, Kent.

1847 Owen, Sir Richard, K.C.B., D.C.L., LL.D., F.R.S., Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, East Sheen, Mortlake.

1873 Stokes, Sir George Gabriel, Bart., M.A., D.C.L., LL.D., F.R.S., M.P., Lucasian Professor of Mathematics in the University of Cambridge; Lensfield Cottage, Cambridge.

Elected

1887 TURNER, SIR WILLIAM, D.C.L., LL.D., F.R.S., Professor of Anatomy in the University of Edinburgh; 6, Eton Terrace, Edinburgh.

1868 TYNDALL, JOHN, D.C.L., LL.D., F.R.S., Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Hindhead House, Hindhead, Surrey.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1878 Baccelli, Guido, M.D., Professor of Medicine at Rome.

1887 Billings, John S., M.D., D.C.L. Oxon., Surgeon U.S.Army; Librarian, Surgeon-General's Office, Washington.

1876 Billroth, Theodor, M.D., Professor of Surgery in the University of Vienna; 20, Alger Strasse, Vienna.

1883 Charcot, J. M., M.D., Physician to the Hôpital de la Salpêtrière, and Professor at the Faculty of Medicine of Paris; Member of the Academy of Medicine; Quai Malaquais 17, Paris.

1883 DuBois Reymond, Emil, M.D., Professor in Berlin; N. W. Neue Wilhelmstrasse 15, Berlin.

1887 Esmarch, Friedrich, M.D., Professor of Surgery in the University of Kiel.

1866 Hannover, Adolph, M.D., Professor at Copenhagen.

1873 von Helmholtz, Hermann Ludwig Ferdinand, Professor of Physics and Physiological Optics; Berlin.

1873 Hofmann, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.

1868 Kölliker, Albert, Professor of Anatomy in the University of Würzburg.

1868 Larrey, Hippolyte Baron, Member of the Institute of France; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

Elected

1883 Pasteur, Louis, LL.D., Member of the Institute of France (Academy of Sciences).

1878 Scanzoni, Friedreich Wilhelm von, Royal Bavarian Privy Councillor ; Professor of Midwifery in the University of Würzburg.

1856 von Virchow, Rudolph, M.D., LL.D., Professor of Pathological Anatomy in the University of Berlin ; Corresponding Member of the Academy of Sciences of the Institute of France ; 10, Schellingstrasse, Berlin.

FELLOWS

OF THE

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P.—President.	V.P.—Vice-President.
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L.—Librarian.	C.—Member of Council.

The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Referee, Sci. Com.*, and *Lib. Com.*, with the dates of office, are attached to the names of those who have served on the Committees of the Society.

SEPTEMBER, 1891.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1846 *Abercrombie, John, M.D.

1877 †Abercrombie, John, M.D., Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square. *Trans.* 1.

1885 Abraham, Phineas S., M.A., M.D., Lecturer on Physiology and Histology at the Westminster Hospital; 2, Henrietta street, Cavendish square.

1851 *Acland, Sir Henry Wentworth, Bart., K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine in the University of Oxford; Broad street, Oxford.

Elected

1885 ACLAND, THEODORE DYKE, M.D., Assistant Physician to St. Thomas's Hospital and to the Hospital for Consumption and Diseases of the Chest, Brompton ; 74, Brook street, Hanover square.

1852 †ADAMS, WILLIAM, Surgeon to the Great Northern Hospital and to the National Hospital for the Paralysed and Epileptic ; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde Park.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, A.M., M.D., LL.D.Glasgow, F.R.S., Commissioner in Lunacy ; Consulting Physician to the Leeds General Infirmary; 3, Melbury Road, Kensington. *Trans.* 3.

1879 ALLCHIN, WILLIAM HENRY, M.B., F.R.S. Ed., Physician to, and Lecturer on Medicine at, the Westminster Hospital; 5, Chandos street, Cavendish square.

1890 ALLINGHAM, HERBERT WILLIAM, 25, Grosvenor street, Grosvenor square.

1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park ; 48, Harley street, Cavendish square. *Trans.* 2.

1884 ANDERSON, ALEXANDER RICHARD, Surgeon to the General Hospital, 5, East Circus Street, Nottingham.

1881 ANDERSON, JAMES, A.M., M.D., Assistant Physician to the London Hospital and to the National Hospital for the Paralysed and Epileptic ; 41, Wimpole street, Cavendish square.

1888 ANDERSON, JOHN, M.D., C.I.E., Physician to the Seamen's Hospital, Greenwich ; 105, Gloucester place, Portman square.

1890 ANDERSON, WILLIAM, Professor of Anatomy to the Royal Academy of Arts ; 2, Harley street, Cavendish square.

Elected

1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. S. 1878-9. C. 1881-2. V.P. 1888. *Trans.* 1.

1891 ANDREWES, FREDERICK WILLIAM, M.B., 15, Upper Brook street.

1891 ANDREWS, LAUNCELOT, M.B., 45, High street, Stamford, Lincolnshire.

1880 *APPLETON, HENRY, M.D. (Travelling),

1888 ARKLE, CHARLES JOSEPH, M.B.

1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

1851 *BAKER, ALFRED, Consulting Surgeon to the Birmingham General Hospital; The Bracken, Augustus road, Edgbaston.

1891 BAKER, CHARLES ERNEST, M.B., St. Bartholomew's Hospital.

1873 *BAKER, J. WRIGHT, Consulting Surgeon to the Derbyshire General Infirmary; 101A, Friar gate, Derby.

1865 BAKER, WILLIAM MORRANT, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 26, Wimpole street, Cavendish square. C. 1878-9. V.P. 1889. *Referee*, 1886-8. *Lib. Com.* 1876-7. *Trans.* 7.

1891 BALGARNIE, WILFRED, M.B., 47, South Molton street.

1887 BALL, JAMES BARRY, M.D., 54, Wimpole street, Cavendish square.

1885 BALLANCE, CHARLES ALFRED, M.S., Senior Assistant Surgeon, West London Hospital; Assistant Surgeon, Hospital for Sick Children, Great Ormond street; Assistant Surgeon for Skin Diseases, St. Thomas's Hospital; 56, Harley street, Cavendish square. *Trans.* 1.

1848 †BALLARD, EDWARD, M.D., F.R.S., Inspector, Medical Department, Local Government Board; 20, Curzon park, Chester. C. 1872. V.P. 1875-6. *Sci. Com.* 1889—. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

Elected

1866 *Banks, Sir John Thomas, M.D., K.C.B., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Queen's University in Ireland; 45, Merrion square, Dublin.

1886 Banks, William Mitchell, M.D., Surgeon to the Liverpool Royal Infirmary; 28, Rodney street, Liverpool.

1879 Barker, Arthur Edward James, Surgeon to University College Hospital, and Assistant Professor of Clinical Surgery and Teacher of Practical Surgery at University College, London; 87, Harley street, Cavendish square. *Trans.* 7.

1882 Barker, Frederick Charles, M.D., Surgeon-Major, Bombay Medical Service [care of Arthur E. J. Barker, 87, Harley street].

1876 Barlow, Thomas, M.D., B.S., Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond street; 10, Wimpole street, Cavendish square. *Trans.* 2.

1881 *Barnes, Henry, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

1861 Barnes, Robert, M.D., Lyss, Hants. C. 1877-8. V.P. 1889–90. *Referee*, 1867-76, 1891. *Lib. Com.* 1869-73. *Sci. Com.* 1889—. *Trans.* 4.

1864 Barratt, Joseph Gillman, M.D.

1880 Barrow, A. Boyce, Assistant Surgeon to King's College Hospital and to the Westminster Hospital; 37, Wimpole street, Cavendish square.

1840 Barrow, Benjamin, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859 Barwell, Richard, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 11.

Elected

1868 Bastian, Henry Charlton, M.A., M.D., F.R.S., Professor of Medicine in University College, London; Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic; 8a, Manchester square. C. 1885. *Referee*, 1886-91. *Trans.* 2.

1890 Bateman. William A. F., Rothesay House, Richmond, Surrey.

1875 Beach, Fletcher, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.

1883 Beale, Edwin Clifford, M.A., M.B., Assistant Physician to the City of London Hospital for Diseases of the Chest; and Physician to the Great Northern Hospital; 23, Upper Berkeley street.

1862 Beale, Lionel Smith, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Referee*, 1873-5. *Trans.* 1.

1860 *Bealey, Adam, M.D., M.A., Oak Lea, Harrogate.

1856 Beardsley, Amos, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1871 Beck, Marcus, M.S., Professor of Surgery in University College, London, and Surgeon to University College Hospital; 30, Wimpole street, Cavendish square. C. 1886-7. *Referee*, 1882-5, 1889-91. *Lib. Com.* 1881-5.

1880 Beevor, Charles Edward, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 33, Harley street, Cavendish square. *Trans.* 1.

1880 Bennett, Alex. Hughes, M.D., Physician to the Westminster Hospital; 76, Wimpole street, Cavendish square. *Trans.* 1.

1883 Bennett, Storer, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to the Dental Hospital of London; 17 George street, Hanover square.

Elected

1877 BENNETT, WILLIAM HENRY, Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 1, Chesterfield street, Mayfair. *Trans.* 4.

1889 BENTLEY, ARTHUR J. M., M.D., 9, Somers place, Hyde Park.

1890 BERRY, DAVID ANDERSON, M.B., C.M., 117, Goldhawk Road.

1845 †BERRY, EDWARD UNWIN, 17, Sherriff road, West Hampstead.

1885 BERRY, JAMES, Assistant Demonstrator of Anatomy, St. Bartholomew's Hospital; 60, Welbeck street, Cavendish square.

1820 BERTIN, STEPHEN, Paris.

1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 54, Prince of Wales road, Norwich.

1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.

1878 BINDON, WILLIAM JOHN VEREKER, M.D., 48, St. Ann's street, Manchester.

1856 †BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

1849 †BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; Westbourne Rectory, Emsworth, Hampshire. C. 1865-6. *Referee*, 1851-9.

1851 †BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 62, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-3. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

Elected

1866 Bishop, Edward, M.D.

1881 Biss, Cecil Yates, M.D., Assistant Physician to, and Lecturer on Materia Medica at, the Middlesex Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 135, Harley street, Cavendish square. *Trans.* 2.

1865 Blanchet, Hilarion, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 Blandford, George Fielding, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 48, Wimpole street, Cavendish square. C. 1883-4.

1846 †Bostock, John Ashton, C.B., *Treasurer;* Hon. Surgeon to H.M. the Queen; Deputy Surgeon-General; 73, Onslow gardens, Brompton. C. 1861-2. V.P. 1870-71. T. 1888-91. *Sci. Com.* 1867.

1890 Bostock, R. Ashton, 73, Onslow gardens, Brompton [Victoria Barracks, Windsor].

1869 Bourne, Walter, M.D. [care of the National Bank of India, 80, King William street, City]; Arcachon, France.

1882 Bowlby, Anthony Alfred, Surgical Registrar to St. Bartholomew's Hospital; 75, Warrington crescent, Maida hill. *Trans.* 4.

1870 *Bowles, Robert Leamon, M.D., 8, West terrace, Folkestone. *Trans.* 1.

1841 †Bowman, Sir William, Bart., LL.D., F.R.S., F.L.S., Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Referee*, 1845-50, 1854-6. *Lib. Com.* 1847. *Trans.* 3.

1886 Boxall, Robert, M.D., Assistant Obstetric Physician to the Middlesex Hospital; 29, Weymouth street, Portland place.

1884 Boyd, Stanley, M.B., Assistant Surgeon to, and Demonstrator of Anatomy at, Charing Cross Hospital; 134, Harley street, Cavendish square.

Elected

1862 Brace, William Henry, M.D., 7, Queen's Gate terrace, Kensington.

1890 Bradford, John Rose, M.D., D.Sc., Assistant Physician to University College Hospital; 52, Upper Berkeley street, Portman square.

1874 Bradshaw, A. F., Surgeon-Major; Surgeon to the Rt. Hon the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]

1883 Bradshaw, James Dixon, M.B., 30, George street, Hanover square.

1867 *Brett, Alfred T., M.D., Watford, Herts.

1876 Bridges, Robert, M.B., Manor House, Yattendon, Newbury, Berks.

1867 Bridgewater, Thomas, M.B., Harrow-on-the-Hill, Middlesex.

1890 Brinton, Roland Danvers, M.D., 8, Queen's Gate terrace.

1868 Broadbent, William Henry, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; 34, Seymour street, Portman square. C. 1885. *Referee*, 1881-4, 1891. *Trans.* 5.

1851 †Brodhurst, Bernard Edward, Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1872 Brodie, George Bernard, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 3, Chesterfield street, Mayfair. *Trans.* 1.

1891 Brodie, Charles Gordon, 30, Harley street, Cavendish square.

1860 Brown-Séquard, Charles Edouard, M.D., LL.D., F.R.S., Laureate of the Academy of Sciences of Paris; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Paris. *Sci. Com.* 1862.

1888 Browne, Henry Langley, Moor House, West Bromwich.

Elected

1878 Browne, Sir James Crichton, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; 7, Cumberland terrace, Regent's Park.

1880 Browne, James William, M.B., 7, Norland place, Holland Park.

1881 Browne, John Walton, M.D., Surgeon to the Belfast Ophthalmological Hospital; 10, College square N., Belfast.

1881 Browne, Oswald Auchinleck, M.A., M.B., Physician to the Royal Hospital for Diseases of the Chest; 43, Bedford square.

1874 Bruce, John Mitchell, M.D., Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 70, Harley street. *Sci. Com.* 1889—. *Referee*, 1886-91. *Lib. Com.* 1888-91. *Trans.* 1.

1871 Brunton, Thomas Lauder, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888-9. *Referee*, 1880-87. *Lib. Com.* 1882-7.

1860 Bryant, Thomas, Consulting Surgeon to Guy's Hospital; Corresponding Member "Société de Chirurgie, Paris;" 65, Grosvenor street, Grosvenor square. C. 1873-4. V. P. 1885-6. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 11. *Pro.* 1.

1864 Buchanan, George, M.D., F.R.S., Medical Officer of the Local Government Board; Member of the Senate of the University of London; 27, Woburn square.

1864 Buckle, Fleetwood, M.D.

1889 Bull, William Charles, M.B., 35, Clarges street, Piccadilly.

1881 Buller, Audley Cecil, M.D.

1885 Butler-Smythe, Albert Charles, Senior Surgeon to the Grosvenor Hospital for Women and Children; 76, Brook street, Grosvenor square.

Elected

1873 Butlin, Henry Trentham, Assistant Surgeon to, and Demonstrator of Practical Surgery and of Diseases of the Larynx at, St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887–8. *Trans.* 3.

1871 Butt, William F.

1883 Buxton, Dudley Wilmot, M.D., B.S., Administrator, and Teacher of the Use, of Anæsthetics, in University College Hospital; Anæsthetist to the Hospital for Women, Soho Square, and to the London Dental Hospital; 82, Mortimer street, Cavendish square.

1868 Buzzard, Thomas, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square. C. 1885-6. *Referee*, 1887-91.

1851 *Cadge, William, Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.

1890 Cagney, James, M.A., M.D., in charge of Electrical Department, St. Mary's Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 11, Welbeck street, Cavendish square. *Trans.* 1.

1885 Cahill, John, 12, Seville street, Lowndes square.

1887 Calvert, James, M.D., 36, Queen Anne street, Cavendish square.

1888 Carless, Albert, M.B., M.S., Assistant Surgeon to King's College Hospital; 15, Stratford place, Oxford street.

1875 Carter, Charles Henry, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde Park.

1853 Carter, Robert Brudenell, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne street, Cavendish square. *Trans.* 1.

1888 Carter, William Jeffreys Becher, Aliwal North, Cape Colony.

1879 Cartwright, S. Hamilton.

1888 Cautley, Edmund, M.B., B.C., 15, Upper Brook street.

1868 Cavafy, John, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. C. 1887. *Lib. Com.* 1888-91. *Trans.* 1.

Elected

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7. *Lib. Com.* 1886-7. *Trans.* 2.

1884 CHAFFEY, WAYLAND CHARLES, M.D., Physician to the Royal Alexandra Hospital for Children; 13, Montpellier road, Brighton.

1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.D., Physician Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; 60, Great Cumberland place. *Referee*, 1891. *Lib. Com.* 1885-91. *Trans.* 7.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

1885 CHAPMAN, PAUL MORGAN, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.

1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; Albert Mansions, 106, Victoria street, Westminster.

1881 *CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; 24, Temple row, Birmingham. *Trans.* 3.

1868 CHEADLE, WALTER BUTLER, M.D., *Trustee;* Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Senior Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. C. 1890-91. *Sci. Com.* 1889—. *Referee*, 1885. *Trans.* 1.

1879 CHEYNE, WILLIAM WATSON, M.B., Surgeon to King's College Hospital, and Demonstrator of Surgery in King's College, London; 59, Welbeck street, Cavendish square. *Lib. Com.* 1886-8, 1891. *Trans.* 1.

1890 CHILDS, CHRISTOPHER, M.D., 2, Royal terrace, Weymouth.

1873 *CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

Elected

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital; 63, Grosvenor street, Grosvenor square. C. 1881-2. *Referee*, 1873-80.

1872 CHRISTIE, THOMAS BEITH, M.D., C.I.E., Medical Superintendent, Royal India Asylum, Ealing.

1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. *Referee*, 1874-81.

1860 CLARK, SIR ANDREW, Bart., M.D., LL.D., F.R.S., *Trustee*, Consulting Physician to, and Emeritus Professor of Clinical Medicine at, the London Hospital; 16, Cavendish square. C. 1875. V.P. 1888.

1879 CLARK, ANDREW, Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square.

1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; The Thorns, Sevenoaks. S. 1847-9. V.P. 1855-6. *Referee*, 1859-81. *Lib. Com.* 1847. *Trans.* 5.

1882 CLARKE, ERNEST, M.D., B.S., Surgeon to the Central London Ophthalmic Hospital; Surgeon and Ophthalmic Surgeon to the Miller Hospital; 41, Lee terrace, Blackheath, and 112, Harley street.

1890 CLARKE, JAMES JACKSON, M.B., Curator of the Museum and Pathologist, St. Mary's Hospital, Paddington.

1848 †CLARKE, JOHN, M.D. C. 1866.

1888 CLARKE, ROBERT HENRY, M.B., Clarence Lodge, Redhill, Surrey.

1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 46, Harley street, Cavendish square.

1842 †CLAYTON, SIR OSCAR MOORE PASSEY, C.B., C.M.G., Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.

Elected

1879 †Clutton, Henry Hugh, M.A., M.B., Assistant Surgeon to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital; 2, Portland place.

1857 Coates, Charles, M.D., Consulting Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.

1888 Cock, Frederick William, M.D., 1, Porchester Houses, Porchester square.

1868 Cockle, John, M.D., F.L.S., Consulting Physician to the Royal Free Hospital; 5, Suffolk place, Pall Mall. *Trans.* 2.

1885 Collins, William Maunsell, M.D., 10, Cadogan place.

1891 Cook, Herbert George, M.B., 162, Cromwell road, South Kensington.

1865 Cooper, Alfred, Consulting Surgeon to the West London Hospital; Surgeon to the Lock Hospital and to St. Mark's Hospital; 9, Henrietta street, Cavendish square.

1868 Cornish, William Robert, C.I.E., late Surgeon-General, Madras Army; Hon. Physician to H.M. the Queen; 8, Cresswell gardens, The Boltons.

1860 *Corry, Thomas Charles Steuart, M.D., Ormean terrace, Belfast.

1889 Cosens, Charles Henry, 49, Oxford terrace, Hyde Park.

1890 Cotes, Charles Edward Henry, M.B., 42, Davies street, Berkeley square.

1860 †Couper, John, Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876. *Referee*, 1882-3.

1877 Coupland, Sidney, M.D., Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; 16, Queen Anne street, Cavendish square.

1862 †Cowell, George, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 3, Cavendish place, Cavendish square. C. 1882-3.

Elected

1868 Crawford, Sir Thomas, K.C.B., M.D., Hon. Surgeon to H.M. the Queen ; late Director-General, Army Medical Department ; 5, St. John's Park, Blackheath. C. 1887.

1869 *Cresswell, Pearson R., Dowlais, Merthyr Tydvil.

1874 Cripps, William Harrison, Assistant Surgeon to St. Bartholomew's Hospital ; 2, Stratford place, Oxford street. C. 1890-91. *Trans.* 1.

1882 Crocker, Henry Radcliffe, M.D., Physician to the Skin Department, University College Hospital ; Physician to the East London Hospital for Children ; 121, Harley street, Cavendish square. *Trans.* 3.

1868 Croft, John, Surgeon to, and Lecturer on Clinical Surgery at, St. Thomas's Hospital ; 48, Brook street, Grosvenor square. C. 1884. V.P. 1890. *Referee*, 1885-88. *Lib. Com.* 1877-8. *Trans.* 2.

1837 Crookes, John Farrar, 45, Augusta gardens, Folkestone.

1872 Crosse, Thomas William, Surgeon to the Norfolk and Norwich Hospital ; 22, St. Giles's street, Norwich.

1890 Crowle, Thomas Henry Rickard, 3, Campden Hill road, Kensington.

1888 Cullingworth, Charles James, M.D., Obstetric Physician and Lecturer on Midwifery at St. Thomas's Hospital ; 46, Brook street, Grosvenor square.

1879 Cumberbatch, A. Elkin, Aural Surgeon to St. Bartholomew's Hospital, and to the Great Northern Hospital ; 17, Queen Anne street, Cavendish square.

1873 Curnow, John, M.D., Professor of Anatomy in King's College, London, and Physician to King's College Hospital ; 3, George street, Hanover square. *Referee*, 1884-91.

1886 Dakin, William Radford, M.D., Obstetric Physician to St. George's Hospital ; 57, Welbeck street, Cavendish square.

1872 Dalby, Sir William Bartlett, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital ; 18, Savile row. *Trans.* 3.

Elected

1884 Dallaway, Dennis, 5, Duchess street, Portland place.

1877 Darbishire, Samuel Dukinfield, M.D., Physician to the Radcliffe Infirmary, Oxford.

1879 Darwin, Francis, M.B., F.R.S., The Grove, Huntingdon road, Cambridge.

1874 Davidson, Alexander, M.D., Physician to the Liverpool Northern Hospital; 2, Gambier terrace, Liverpool.

1852 Davies, William, M.D., 2, Marlborough buildings, Bath.

1876 Davies-Colley, J. Neville C., M.C., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 2.

1878 Davy, Richard, F.R.S. Ed., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square. *Trans.* 1.

1882 *Dawson, Yelverton, M.D., Heathlands, Southbourne-on-Sea, Hants.

1867 Day, William Henry, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1889 Dean, Henry Percy, M.B., B.S., 60, Gower street.

1889 Delépine, Sheridan, B.S., M.B., 6, Chapel place, Cavendish square. *Trans.* 1.

1878 Dent, Clinton Thomas, Assistant Surgeon to, and Lecturer on Practical Surgery at, St. George's Hospital; 61, Brook street. C. 1890. *Trans.* 4.

1859 †Dickinson, William Howship, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. C. 1874-5. V.P. 1887. *Referee*, 1869-73. 1882-6. *Sci. Com.* 1867, 1879, 1889—. *Trans.* 13.

1891 Dickinson, William Lee, M.B., 9, Chesterfield street, May Fair.

Elected

1839 †Dixon, James, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.

1889 Dodd, Henry Work, 47, Kensington Park gardens.

1845 Dodd, John.

1888 Donelan, James, M.B., M.C., 2, Upper Wimpole street, Cavendish square.

1879 Donkin, Horatio, M.B., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 108, Harley street, Cavendish square.

1877 Doran, Alban Henry Griffiths, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. *Lib. Com.* 1891. *Trans.* 1.

1863 Down, John Langdon Haydon, M.D., Consulting Physician to the London Hospital; 81, Harley street, Cavendish square. C. 1880. V.P. 1890-91. *Trans.* 2.

1867 Drage, Charles, M.D., Hatfield, Herts.

1884 Drage, Lovell, M.B., B.S., The Small House, Hatfield, Herts.

1879 Drewitt, F. G. Dawtrey, M.D., Physician to the West London Hospital and to the Victoria Hospital for Children; 2, Manchester square.

1885 Drummond, David, M.D., 7, Saville Place, Newcastle-on-Tyne.

1880 Drury, Charles Dennis Hill, M.D., Bondgate, Darlington.

1865 Drysdale, Charles Robert, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 23, Sackville street, Piccadilly.

1865 †Duckworth, Sir Dyce, M.D., LL.D., Hon. Physician to H.R.H. the Prince of Wales; Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee* 1885-91. *Trans.* 2.

Elected

1876 Dudley, William Lewis, M.D., Physician to the City Dispensary; 149, Cromwell road, South Kensington.

1874 Duffin, Alfred Baynard, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.

1871 Duke, Benjamin, Windmill House, Clapham Common.

1871 *Dukes, Clement, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.

1867 Dukes, M. Charles, M.D., Wellesley Villa, Wellesley road, Croydon.

1880 Dunbar, James John Macwhirter, M.D., Hedingham House, Clapham Common.

1889 *Duncan, John, M.D., St. Petersburg, Russia.

1884 Duncan, William, M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 6, Harley street, Cavendish square.

1887 Dunn, Hugh Percy, Assistant Ophthalmic Surgeon and Pathologist at the West London Hospital; 39, Welbeck street, Cavendish square.

1863 Durham, Arthur Edward, F.L.S., Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. V.P. 1887. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.

1874 Durham, Frederic, M.B., 82, Brook street, Grosvenor square.

1843 Durrant, Christopher Mercer, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.

1872 Eager, Reginald, M.D., Northwoods, near Bristol.

1887 Easmon, John Farrell, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony; Accra, Gold Coast, West Africa.

Elected

1868 EASTES, GEORGE, M.B.Lond., 35, Gloucester place, Hyde Park.

1888 ECCLES, ARTHUR SYMONS, M.B., C.M., 23, Hertford street, May Fair.

1891 EDDOWES, ALFRED, M.D., 25, Old Burlington street.

1883 EDMUNDS, WALTER, M.C., 75, Lambeth Palace road, Albert Embankment. *Trans.* 2.

1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital, and to St. Peter's Hospital for Stone; 55, Harley street, Cavendish square.

1824 EDWARDS, GEORGE.

1891 ELAM, GEORGE, 75, Harley street, Cavendish square.

1887 ELLIOTT, JOHN, Whitefriars Lodge, Chester.

1848 ELLIS, GEORGE VINER, Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.

1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1889 ELLISTON, WILLIAM ALFRED, M.D., Manor House, Ipswich.

1842 †ERICHSEN, JOHN ERIC, LL.D.,F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish sq. C.1855-6. V.P. 1868. P.1879-80. *Referee*, 1866-8, 1884-89. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1879 EVE, FREDERIC S., Assistant Surgeon to the London Hospital; Surgeon to Out-Patients at the Evelina Hospital for Sick Children; 125, Harley street, Cavendish square. *Trans.* 2.

1877 EWART, WILLIAM, M.D., Physician to St. George's Hospital; 33, Curzon street,Mayfair. *Sci. Com.* 1889—. *Trans.* 1.

1875 *FAGAN, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital; 1, Glengall place, Belfast.

Elected

1869 Fairbank, Frederick Royston, M.D., 46, Hallgate, Doncaster.

1862 Farquharson, Robert, M.D., LL.D.Aber., M.P., Migvie Lodge, Porchester gardens, Hyde Park (Finzean, Aboyne, Aberdeenshire). *Lib. Com.* 1876-80.

1872 Fayrer, Sir Joseph, K.C.S.I., M.D., F.R.S., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Surgeon-General, India Office; Physician to the Secretary of State for India in Council; President of the Indian Medical Board; 53, Wimpole street, Cavendish square. C. 1888. *Referee*, 1881-7.

1887 Feeny, Michael Henry, Les Avants, Montreux, Switzerland.

1872 *Fenwick, John C. J., M.D., Physician to the Durham County Hospital; 25, North road, Durham.

1863 Fenwick, Samuel, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882-91. *Trans.* 4.

1880 Ferrier, David, M.D., LL.D., F.R.S., Professor of Forensic Medicine in King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Trans.* 2.

1852 *Field, Alfred George.

1889 Field, George P., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital; 34, Wimpole street, Cavendish square.

1879 Finlay, David White, M.D., Professor of the Practice of Medicine in the University of Aberdeen. *Trans.* 2.

1866 Fitz-Patrick, Thomas, A.M., M.D., 30, Sussex gardens, Hyde Park.

1891 Fletcher, Herbert Morley, M.B., St. Bartholomew's Hospital.

1842 Fletcher, Thomas Bell Elcock, M.D., Consulting Physician to the Birmingham General Hospital; 8, Clarendon crescent, Leamington. *Trans.* 1.

Elected

1864 *Folker, William Henry, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1877 de Fonmartin, Henry, M.D., 1, Anchor Gate terrace, Portsea, Hants.

1865 Foster, Sir Balthazar Walter, M.D., M.P., Professor of Medicine at the Queen's College, Birmingham, and Consulting Physician to the Birmingham General Hospital; 55, Temple row, Birmingham.

1883 Fowler, James Kingston, M.A., M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

1859 Fox, Edward Long, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.

1887 Fox, Richard Hingston, M.D., 23, Finsbury square.

1880 Fox, Thomas Colcott, B.A., M.B., Physician to the Skin Department of the Paddington Green Hospital for Children, and Assistant Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

1871 Frank, Philip, M.D., Cannes, France.

1884 *Franks, Kendal, M.D., Surgeon to the Adelaide Hospital and to the Throat and Ear Hospital, Dublin; 69, Fitzwilliam square, Dublin. *Trans.* 1.

1843 Fraser, Patrick, M.D., C. 1866.

1889 Freeman, Henry William, 24, The Circus, Bath.

1868 Freeman, William Henry, 21, St. George's square, South Belgravia.

1884 Fuller, Charles Chinner, 10, St. Andrew's place, Regent's Park.

1883 Fuller, Henry Roxburgh, M.D., 45, Curzon street, May Fair.

1876 Furner, Willoughby, Assistant Surgeon to the Sussex County Hospital; 2, Brunswick place, Brighton.

Elected

1864 *Gairdner, William Tennant, M.D., LL.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow. *Trans.* 1.

1874 †Galabin, Alfred Lewis, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; 49, Wimpole st., Cavendish square. *Referee*, 1882-91. *Lib. Com.* 1883-4. *Trans.* 2.

1883 Galton, John Charles, M.A., F.L.S., 45, Great Marlborough street.

1885 Gamgee, Arthur, M.D., F.R.S., 14, Panton street, Cambridge.

1865 Gant, Frederick James, Consulting Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. *Referee*, 1886-91. *Lib. Com.* 1882-5. *Trans.* 3.

1867 Garland, Edward Charles, Yeovil, Somerset.

1867 Garlike, Thomas W., Malvern Cottage, Churchfield road, Ealing.

1854 †Garrod, Sir Alfred Baring, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.

1886 Garrod, Archibald Edward, M.A., M.D., Assistant Physician to the West London Hospital; 9, Chandos street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 3.

1879 Garstang, Thomas Walter Harropp, Headingley House, Knutsford, Cheshire.

1889 *Gaskell, Walter Holbrook, M.D., F.R.S., Lecturer on Physiology, University of Cambridge; Petersfield House, Parkside, Cambridge.

1819 Gaulter, Henry.

1887 Gay, John, 119, Upper Richmond road, Putney.

Elected

1866 GEE, SAMUEL JONES, M.D., *Librarian,* Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Consulting Physician to the Hospital for Sick Children; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887-91. *Sci. Com.* 1879. *Referee,* 1885-7. *Lib. Com.* 1871-6. *Trans.* 1.

1885 GELL, HENRY WILLINGHAM, M.B., 43, Albion street, Hyde Park.

1878 GERVIS, HENRY, M.D., Consulting Obstetric Physician to St. Thomas's Hospital; Consulting Physician to the Royal Maternity Charity; 40, Harley street, Cavendish square. *Referee,* 1884-91. *Trans.* 1.

1884 GIBBES, HENEAGE, M.D., Professor of Pathology in the University of Michigan; Ann Arbor, Michigan, U.S.A.

1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place.

1877 GODLEE, RICKMAN JOHN, Surgeon to University College Hospital, and Teacher of Operative Surgery in University College, London; Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Consumption, Brompton; 81, Wimpole street, Cavendish square. *Referee,* 1886-91. *Trans.* 6.

1870 †GODSON, CLEMENT, M.D., Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.

1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Assistant Surgeon and Lecturer on Physiology at Guy's Hospital; 12 Queen Anne street, Cavendish square. *Trans.* 1.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Referee,* 1860-3. *Lib. Com.* 1863. *Trans.* 2.

1883 GOODHART, JAMES FREDERIC, M.D., Physician to Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 25, Weymouth street, Portland place.

Elected

1889 GOODSALL, DAVID HENRY, 17, Devonshire place, Upper Wimpole street.

1890 †GORDON, WILLIAM, M.B., Barnfield Lodge, Exeter.

1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to the Middlesex Hospital; 10, Queen Anne street, Cavendish square. *Lib. Com.* 1891. *Trans.* 2.

1873 GOWERS, WILLIAM RICHARD, M.D., F.R.S., Consulting Physician to University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. C. 1891. *Referee* 1888-90. *Lib. Com.* 1884-6. *Trans.* 7.

1851 †GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury square.

1868 GREEN, T. HENRY, M.D., Physician to Charing Cross Hospital, and to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee,* 1882-5.

1889 GREENE, GEORGE EDWARD JOSEPH, "The Dell," Ballycarney Ferns, County Wexford.

1875 *GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee,* 1881.

1882 GRESSWELL, DAN ASTLEY, M.B., Melbourne, Victoria.

1885 GRIFFITH, WALTER SPENCER ANDERSON, M.B., Physician to the Samaritan Free Hospital for Women and Children; 114, Harley street, Cavendish square.

1889 GRIFFITHS, JOSEPH, M.B., C.M., 17, Fitzwilliam street, Cambridge.

1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; 27, Curzon street, Mayfair.

1852 GROVE, JOHN, Fyning, 15, Johnstown street, Bath.

Elected

1889 Gubb, Alfred Samuel, M.D.Paris ; 29, Gower street.

1860 Gueneau de Mussy, Henri, M.D. ; 15, Rue du Cirque, Paris. *Lib. Com.* 1863-5.

1883 Gunn, Robert Marcus, M.B., Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields ; Ophthalmic Surgeon to the Hospital for Sick Children, Great Ormond Street ; 54, Queen Anne street, Cavendish square.

1890 Guthrie, Leonard George, M.B., B.S., 24, Upper George street, Bryanston square.

1886 Habershon, Samuel Herbert, M.D., 70, Brook street, Grosvenor square.

1888 Hadden, Walter Baugh, M.D., Assistant Physician and Lecturer on Materia Medica at St. Thomas's Hospital ; Assistant Physician, Hospital for Sick Children ; 21, Welbeck street, Cavendish square.

1885 Haig, Alexander, M.D., Physician to the Royal Hospital for Children and Women ; 7, Brook street, Grosvenor square. *Trans.* 5.

1890 Hale, Charles Douglas Bowdich, M.D., 8, Sussex gardens, Hyde Park.

1881 Hall, Francis de Havilland, M.D., Assistant Physician, and Physician to the Throat Department, and Lecturer on Practical Medicine at the Westminster Hospital ; Physician to St. Mark's Hospital ; 47, Wimpole street, Cavendish square.

1885 Halliburton, William Dobinson, M.D., F.R.S., Professor of Physiology, King's College, London ; 9, Ridgmount gardens, Gower street.

1891 Hamer, William Heaton, M.B., Ladywell, Dartmouth Park Hill, Highgate.

1870 Hamilton, Robert, Surgeon to the Royal Southern Hospital, Liverpool ; 1 Prince's road, Liverpool.

1889 Handfield-Jones, Montagu, M.D., Assistant Obstetric Physician to St. Mary's Hospital ; 35, Cavendish square.

Elected

1874 Hardie, Gordon Kenmure, M.D., Deputy Inspector General of Hospitals; Florence road, Ealing.

1856 †Hare, Charles John, M.D., *Treasurer*, late Professor of Clinical Medicine in University College, London, and Consulting Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4. T. 1887-91.

1857 Harley, George, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. V.P. 1891. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3. *Trans.* 1.

1864 Harley, John, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882-91. *Sci. Com.* 1879. *Trans.* 10.

1880 Harris, Vincent Dormer, M.D., Physician to the Victoria Park Hospital; Demonstrator of Physiology at St. Bartholomew's Hospital; 31, Wimpole street, Cavendish square.

1870 Harrison, Reginald, 6, Lower Berkeley Street, Portman square. *Trans.* 1.

1854 Haviland, Alfred.

1890 Haviland, Frank Papillon, M.B., B.C., 57, Warrior square, St. Leonard's-on-Sea.

1870 Haward, J. Warrington, *Secretary;* Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 16, Savile row, Burlington Gardens. C. 1885. S. 1888-91. *Lib. Com.* 1881-4. *Sci. Com.* 1889—. *Trans.* 2.

1838 †Hawkins, Charles, Inspector of Anatomical Schools in London; 9, Duke street, Portland place. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Referee*, 1859-60. *Lib. Com.* 1843. *Trans.* 2.

1885 Hawkins, Francis Henry, M.B., 59, Wimpole street, Cavendish square.

Elected

1891 Hawkins, Herbert Pennell, M.B., B.C., Assistant Physician to St. Thomas's Hospital; 38, Weymouth street, Portland place.

1848 †Hawksley, Thomas, M.D., 11, Albert Mansions, Victoria street, and Beomands, Chertsey, Surrey.

1875 Hayes, Thomas Crawford, M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital; 17, Clarges street, Piccadilly.

1860 Hayward, Henry Howard, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

1891 Hayward, John Arthur, M.B., St. Bartholomew's Hospital.

1861 Hayward, William Henry.

1865 Heath, Christopher, *Trustee*, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. V.P. 1889. *Lib. Com.* 1870-3. *Trans.* 3.

1850 Heaton, George, M.D., Boston, U.S.

1882 Hensley, Philip John., M.D., Assistant Physician and Lecturer on Forensic Medicine to St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.

1877 Herman, George Ernest, M.B., Obstetric Physician to and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Trans.* 1.

1877 Heron, George Allan, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.

1891 Herring, Herbert T., M.B., B.S., 50, Harley street, Cavendish square.

1883 Herringham, Wilmot Parker, M.D., 13, Upper Wimpole street, Cavendish square. *Trans.* 1.

1887 Hewitt, Frederic William, M.D., 10, George street, Hanover square. *Trans.* 1.

1855 †Hewitt, W. M. Graily, M.D., Emeritus Professor of Midwifery in University College, London, and Consulting Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Referee*, 1868-75, 1877-91. *Lib. Com.* 1868, 1874.

Elected

1880 Hicks, Charles Cyril, M.D., Wokingham, Berks.

1890 Hickson, Albert T., M.A., M.D., 29, Norland square, Notting Hill.

1873 Higgens, Charles, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 2.

1890 Hill, G. William, M.D., B.Sc., 24, Wimpole street, Cavendish square.

1862 Hill, M. Berkeley, M.B., Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 66, Wimpole street, Cavendish square. *Referee*, 1888-91. C. 1878-9. S. 1881-4. V.P. 1885-6. *Trans.* 1.

1843 †Holden, Luther, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1879 Holland, Philip Alexander, M.A.

1868 Hollis, William Ainslie, M.A., M.D., Assistant-Physician to the Sussex County Hospital; 8, Cambridge road, Brighton.

1856 †Holmes, Timothy, M.A., *President*, Consulting Surgeon to St. George's Hospital; Corresponding Member of the "Société de Chirurgie," Paris; 18, Great Cumberland place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. P. 1890-91. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5. *Trans.* 8.

1846 †Holt, Barnard Wight, Consulting Surgeon to the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington Gardens. C. 1862-3. V.P. 1879-80.

1846 †Holthouse, Carsten, 1, Bath terrace, Richmond. C. 1863. *Referee*, 1870-6. *Lib. Com.* 1859-60.

Elected

1878 Hood, Donald William Charles, M.D., Senior Physician to the North-West London Hospital; Physician to the West London Hospital; 43, Green street, Park lane.

1883 Horsley, Victor Alexander Haden, F.R.S., Assistant Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; Professor of Pathology in University College, London; 25, Cavendish square. *Trans.* 1.

1865 Howard, Benjamin, M.D. [New York, U.S.] *Trans.* 1.

1881 Howard, Henry, M.B., abroad. [6, The Terrace, Mount Pleasant, Cambridge.]

1874 Howse, Henry Greenway, M.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. C. 1890. *Sci. Com.* 1879. *Referee*, 1887-89. *Trans.* 2.

1886 Hudson, Charles Elliott Leopold Barton, Surgical Registrar, Middlesex Hospital; 6, Chandos street, Cavendish square.

1884 Huggard, William R., M.D. [Place de la Synagogue, 2, Genève.]

1857 †Hulke, John Whitaker, F.R.S., *Librarian*, Surgeon to the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879-91. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 9.

1889 Humphery, Francis William, M.A., M.B., Micklem Hall, Oxford.

1855 Humphry, Sir George Murray, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 9.

1882 Humphry, Laurence, M.B., 3, Trinity street, Cambridge.

1889 Hunter, William, M.D., F.R.S.Ed., 61, Wimpole street, Cavendish square.

Elected

1873 Hunter, Sir W. Guyer, M.D., M.P., Hon. Surgeon to H.M. the Queen ; late Principal of, and Professor of Medicine in, Grant Medical College, Bombay ; Surgeon-General Bombay Army ; 21, Norfolk crescent, Hyde park.

1849 Hussey, Edward Law, Consulting Surgeon to the Oxford County Lunatic Asylum and the Warneford Asylum ; 24, Winchester road, Oxford. *Trans.* 1.

1856 †Hutchinson, Jonathan, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital ; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields ; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. *Referee,* 1876-81, 1883-91. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.

1888 Hutchinson, Jonathan, Jun., Assistant Surgeon to the London Hospital ; 12, Old Cavendish street, Cavendish square.

1820 Hutchinson, William, M.D.

1847 Image, William Edmund, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.

1856 Inglis, Cornelius, M.D.

1871 Jackson, J. Hughlings, M.D., F.R.S., Physician to the London Hospital ; Physician to the National Hospital for the Paralysed and Epileptic ; 3, Manchester square. C. 1889.

1841 †Jackson, Paul, 51, Wellington road, St. John's Wood. C. 1862.

1863 Jackson, Thomas Vincent, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital ; Whetstone House, Waterloo road south, Wolverhampton.

1883 Jacobson, Walter Hamilton Acland, B.A., M.B., M.S., Assistant Surgeon and Lecturer on Anatomy to Guy's Hospital ; Surgeon to the Royal Hospital for Children and Women ; 66, Great Cumberland place, Hyde Park. *Trans.* 2.

1825 James, John B., M.D.

Elected

1883 *Jenkins, Edward Johnstone, M.D., The Australian Club, Sydney, New South Wales.

1851 †Jenner, Sir William, Bart., M.D., K.G.C.B., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Member of the Senate of the University of London; Greenwood, Bishop's Waltham, Hants. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

1884 Jennings, Charles Egerton, M.S., M.B.

1881 Jennings, William Oscar, M.D., 35, Rue Marbœuf, Avenue des Champs-Elysées, Paris.

1884 Jessett, Frederic Bowreman, Surgeon to the Royal General Dispensary; 16, Upper Wimpole street.

1883 Jessop, Walter H. H., M.B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 73, Harley street.

1851 Johnson, Edmund Charles, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

1847 †Johnson, George, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. P. 1884-5. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib. Com.* 1860-1. *Trans.* 10. *Pro.* 1.

1881 Johnson, George Lindsay, M.A., M.D., Cortina, Netherhall gardens, South Hampstead, and 14, Stratford place, Oxford street.

1889 Johnson, Harold J., Senior Assistant, Gloucester County Asylum.

1889 Johnson, Raymond, M.B., B.S., Surgeon to Out-Patients at the Great Northern Hospital and the Victoria Hospital for Children; 123, Gower street.

1884 Johnston, James, M.D., 11, Chester place, Hyde Park square.

Elected

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1887 JONES, HENRY LEWIS, M.D., Medical Officer in charge of Electrical Department at St. Bartholomew's Hospital; 9, Upper Wimpole street, Cavendish square.

1876 JONES, LESLIE HUDSON, M.D., Limefield House, Cheetham hill, Manchester.

1875 *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, and Fellow of the Senate, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., Wool Exchange.]

1865 JORDAN, FURNEAUX, Consulting Surgeon to the Queen's Hospital, Birmingham; Selly Hill, Birmingham.

1881 JULER, HENRY EDWARD, Junior Ophthalmic Surgeon to St. Mary's Hospital; 77, Wimpole street, Cavendish square.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square.

1872 KELLY, CHARLES, M.D., Professor of Hygiene in King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District; Ellesmere, Gratwicke road, Worthing, Sussex.

1848 *KENDELL, DANIEL BURTON, M.D., Thornhill House, Walton, near Wakefield, Yorkshire.

1890 *KERR, J. G. DOUGLAS, M.B., C.M., 6, The Circus, Bath.

1884 KESER, JEAN SAMUEL, M.D., Surgeon to the French Hospital, Leicester place; 11, Harley street, Cavendish square.

1877 *KHORY, RUSTONJEE NASERWANJEE, M.D., Physician to the Parell Dispensary, Bombay; Girgaum road, Bombay.

1857 †KIALLMARK, HENRY WALTER, 5, Pembridge gardens, Bayswater. C. 1890-91.

Elected

1881 KIDD, PERCY, M.A., M.D., Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook street, Grosvenor square. *Trans.* 4.

1851 †KINGDON, JOHN ABERNETHY, Consulting Surgeon to the Bank of England; 2, Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1885 KLEIN, EDWARD EMANUEL, M.D., F.R.S., Lecturer on Physiology, St. Bartholomew's Hospital; 19, Earl's Court square.

1883 KNAPTON, GEORGE, 4, Clivedon place, Eaton square.

1888 KYNSEY, WILLIAM RAYMOND, C.M.G., Inspector-General of Hospitals, Colombo, Ceylon.

1889 LANCASTER, ERNEST LE CRONIER, M.B., B.Ch., Demonstrator of Anatomy at St. George's Hospital.

1891 LANE, HUGH, 11, The Circus, Bath.

1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; St. Mary's, Madeley road, Ealing. C. 1849-50. V.P. 1865. *Referee*, 1850.

1884 LANE, WILLIAM ARBUTHNOT, M.S., Assistant Surgeon to Guy's Hospital and to the Hospital for Sick Children; 8, St. Thomas's street, Southwark. *Trans.* 3.

1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 26, Upper Wimpole street, Cavendish square.

1865 LANGTON, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881-2. *Referee*, 1885-91. *Lib. Com.* 1879-80, 1888-91. *Trans.* 2.

1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

Elected

1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1890 LAW, EDWARD, M.D., C.M., 35, Harley street, Cavendish square.

1816 LAWRENCE, G. E.

1890 LAWRENCE, HENRY CRIPPS, 12, Sussex gardens, Hyde Park.

1888 LAWRENCE, LAURIE ASHER, 125, Harley street, Cavendish square.

1890 *LAWRIE, EDWARD, M.B., Indian Medical Department; Hyderabad, Deccan.

1884 LAWSON, GEORGE, Surgeon-Oculist to H.M. the Queen; Surgeon to the Royal London Ophthalmic Hospital and to the Middlesex Hospital; 12, Harley street, Cavendish square.

1880 LAYCOCK, GEORGE LOCKWOOD, M.B., Melbourne, Victoria, Australia.

1886 *LEDIARD, HENRY AMBROSE, M.D., Surgeon to the Cumberland Infirmary; 41, Lowther street, Carlisle.

1882 LEDWICH, EDWARD L'ESTRANGE, Lecturer on Surgical and Descriptive Anatomy in the Ledwich School of Medicine, Dublin; 23, Upper Leeson street, Dublin.

1843 †LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 14. *Pro.* 2.

1884 LEE, ROBERT JAMES, M.D., 6, Savile row, Burlington gardens.

1883 LEESON, JOHN RUDD, M.D., C.M., 6, Clifden road, Twickenham.

1869 LEGG, JOHN WICKHAM, M.D., C. 1886. *Referee*, 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.

1836 LEIGHTON, FREDERICK, M.D.

1886 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., Assistant Obstetric Physician to the London Hospital and Physician to Out-patients of Queen Charlotte's Lying-in Hospital; 60, Wimpole street, Cavendish square.

Elected

1872 LIEBREICH, RICHARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; Paris.

1878 LISTER, SIR JOSEPH, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery at King's College, London; and Surgeon to King's College Hospital; 12, Park crescent, Regent's Park.

1872 *LITTLE, DAVID, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; 21, St. John street, Manchester.

1891 LITTLE, ERNEST MUIRHEAD, 18, Park street, Grosvenor square.

1889 *LITTLE, JAMES, M.D., Physician to the Adelaide Hospital; 14, Stephen's Green North, Dublin.

1889 LITTLE, JOHN FLETCHER, M.B., 60, Welbeck street, Cavendish square.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1819 LLOYD, ROBERT, M.D.

1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Central Hospital, and Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital; 19, Upper Berkeley street. *Trans.* 2.

1860 LONGMORE, SIR THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, Foreign Associate "Académie de Médecine;" Woolston Lawn, Woolston, Hants. *Trans.* 2.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Belmont, Wateringbury, Kent.

1881 LUCAS, RICHARD CLEMENT, B.S., M.B., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury square. *Trans.* 1.

1888 LUFF, ARTHUR PEARSON, M.D., B.Sc., 47, Weymouth street, Portland place.

Elected

1883 Lund, Edward, Professor of Surgery, and Member of Senate, Victoria University, Manchester; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.

1887 Lush, Percy J. F., M.B., 8, Fitzjohn's avenue, South Hampstead.

1867 Maberly, George Frederick, Mailai Valley, Nelson, New Zealand.

1889 MacAlister, Donald, M.A., B.Sc., M.D., Physician to Addenbrooke's Hospital; Lecturer on Medicine, St. John's College; University Lecturer in Medicine; St. John's College, Cambridge.

1873 †MacCarthy, Jeremiah, M.A., Surgeon to the London Hospital and Lecturer on Physiology at the London Hospital Medical College; 15, Finsbury square. C. 1886-7. *Lib. Com.* 1882-5. *Referee*, 1890-91.

1867 Mac Cormac, Sir William, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. C. 1884-5. *Referee*, 1889-91. *Trans.* 1.

1887 Macdonald, George Childs, M.D.

1880 Macfarlane, Alexander William, M.D., Examiner in Medical Jurisprudence, University of Glasgow; 6, Manchester square.

1866 Macgowan, Alexander Thorburn, M.D.

1880 McHardy, Malcolm Macdonald, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Surgeon to the Royal South London Ophthalmic Hospital; 5, Savile row.

1822 Macintosh, Richard, M.D.

1859 *M'Intyre, John, M.D., LL.D., Odiham, Hants.

1873 MacKellar, Alexander Oberlin, M.S.I., Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 79, Wimpole street, Cavendish square.

Elected

1881 MACKENZIE, STEPHEN, M.D., Physician to the London Hospital, and Lecturer on the Principles and Practice of Medicine at the London Hospital Medical College; Physician to the Royal London Ophthalmic Hospital; 18, Cavendish square. *Referee*, 1890-91. *Trans.* 1.

1885 MACKERN, JOHN, M.D., St. Germain's Lodge, Shooter's Hill road, Blackheath.

1876 MACKEY, EDWARD, M.D., Assistant Physician to the Sussex County Hospital; 1, Brunswick road, Hove, Brighton.

1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1879 MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.

1889 MACLEHOSE, NORMAN MACMILLAN, M.B., C.M., 13, Queen Anne street, Cavendish square.

1876 MACNAMARA, CHARLES N., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon-Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street. C. 1891. *Referee*, 1884-90. *Lib. Com.* 1886-90.

1881 MACREADY, JONATHAN FORSTER CHRISTIAN HORACE, Surgeon to the Great Northern Hospital; 51, Queen Anne street, Cavendish square.

1880 MADDICK, EDMUND DISTIN, 2, Chandos street, Cavendish square.

1886 MAGUIRE, ROBERT, M.D., 4, Seymour street, Portman square. *Sci. Com.* 1889—.

1880 MAKINS, GEORGE HENRY, Assistant Surgeon to St. Thomas's Hospital and Surgeon to the Evelina Hospital for Children; 2, Queen street, May Fair. *Trans.* 1.

1885 MALCOLM, JOHN DAVID, M.B., Surgeon in charge of Out-Patients, Samaritan Free Hospital; 24, Bryanston street, Portman square. *Trans.* 1.

Elected

1891 MANBY, ALAN REEVE, M.D., Surgeon Apothecary to their Royal Highnesses the Prince and Princess of Wales at Sandringham; East Rudham, Norfolk.

1890 MANSON, PATRICK, M.D., C.M., 21, Queen Anne street, Cavendish square.

1888 MAPOTHER, EDWARD DILLON, M.D., 32, Cavendish square.

1855 MARCET, WILLIAM, M.D., F.R.S., Flowermead, Wimbledon Park, and Athenæum Club, Pall Mall. C. 1871. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

1867 MARSH, F. HOWARD, Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 30, Bruton street, Berkeley square. C. 1882-3, 1889. S. 1885-7. V.P. 1891. *Lib. Com.* 1880-1. *Trans.* 4.

1891 MARTIN, HENRY CHARRINGTON, M.D., 11, Somers place, Hyde Park.

1884 MARTIN, SIDNEY HARRIS COX, M.D., Assistant Physician to University College Hospital; 10, Mansfield street, Portland place.

1883 MAUDSLEY, HENRY CARR, M.D., 11, Spring street, Melbourne, Victoria.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1865 MEDWIN, AARON GEORGE, M.D., Consulting Dental Surgeon to the Royal Kent Dispensary, 34, Bruton street, Berkeley square.

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 6, Queen Anne street, Cavendish square. *Trans.* 1.

1874 MERRIMAN, JOHN J., 45, Kensington square.

1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

1885 MILLICAN, KENNETH WILLIAM, B.A.

1882 MILLS, JOSEPH, 28, Queen Anne street, Cavendish square.

Elected

1873 MILNER, EDWARD, Surgeon to the Lock Hospital; 32, New Cavendish street, Portland place.

1887 MIVART, FREDERICK ST. GEORGE, M.D., Beaumont Lodge, Worple road, Wimbledon.

1883 MONEY, ANGEL, M.D., Assistant Physician to the Hospital for Sick Children, Great Ormond street; 24, Harley street, Cavendish square. *Trans.* 4.

1873 MOORE, NORMAN, M.D., Assistant Physician and Lecturer on Pathology at St. Bartholomew's Hospital; 94, Gloucester place, Portman square. C. 1891. *Referee*, 1886-90. *Sci. Com.* 1889—.

1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Victoria University, Manchester; 1, St. Peter's square, Manchester.

1878 MORGAN, JOHN HAMMOND, M.A., Surgeon to the Charing Cross Hospital and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. *Trans.* 2.

1891 MORRIS, GRAHAM, Wallington, Surrey.

1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 8, Cavendish square. C. 1888-9. *Referee*, 1882-7. *Trans.* 10.

1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital; 8, Harley street, Cavendish square. *Sci. Com.* 1889—.

1885 MOTT, FREDERICK WALKER, M.D., Lecturer on Physiology, Charing Cross Hospital; 84, Wimpole street, Cavendish square.

1879 MUNK, WILLIAM, M.D., Harveian Librarian, Royal College of Physicians; Consulting Physician to the Royal Hospital for Incurables; 40, Finsbury square.

1888 †MURRAY, HUBERT MONTAGUE, M.D., 27, Savile row, Burlington gardens.

Elected

1873 MURRAY, J. IVOR, M.D., F.R.S.Ed. 24, Huntriss row, Scarborough.

1880 MURRELL, WILLIAM, M.D., Assistant Physician to the Royal Hospital for Diseases of the Chest; Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, the Westminster Hospital; 17, Welbeck street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.

1863 MYERS, ARTHUR BOWEN RICHARDS, Brigade-Surgeon, Brigade of Guards; 43, Gloucester street, Warwick square. C. 1878-9. *Lib. Com.* 1877.

1882 MYERS, ARTHUR THOMAS, M.D., 9, Lower Berkeley street, Portman square.

1889 NAPIER, FRANCIS HORATIO, M.B.

1881 NALL, SAMUEL, M.B.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole street, Cavendish square.

1889 NEVINS, ARTHUR EDWARD, Eastwood place, Hanley, Staffordshire.

1843 †NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., Trenanen, Newquay, Cornwall.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants. *Lib. Com.* 1857.

1847 *NOURSE, WILLIAM EDWARD CHARLES, Bouverie House, Exeter.

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

Elected

1884 Oakes, Arthur, M.D.

1880 O'Connor, Bernard, A.B., M.D., Physician to the North London Hospital for Consumption; Greenhill Park, Harlesden.

1847 O'Connor, Thomas, March, Cambridgeshire.

1880 Ogilvie, George, M.B., Lecturer on Experimental Physics at the Westminster Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 22, Welbeck street, Cavendish square.

1880 Ogilvie, Leslie, M.B., Physician to the Paddington Green Children's Hospital; 46, Welbeck street, Cavendish square.

1891 Ogle, Cyril, M.A., M.B., 30, Cavendish square.

1858 Ogle, John William, M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.

1855 *Ogle, William, M.A., M.D., Physician to the Derbyshire Infirmary; The Elms, Duffield road, Derby.

1860 Ogle, William, M.D., Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.

1870 Oldham, Charles Frederic, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

1883 *Oliver, Thomas, M.D., Lecturer on Practical Physiology, University of Durham; and Physician to the Newcastle-upon-Tyne Infirmary; 12, Eldon square, Newcastle-on-Tyne. *Trans.* 1.

1871 *O'Neill, William, M.D., Physician to the Lincoln Lunatic Hospital, Silver street, Lincoln.

1873 Ord, William Miller, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook street, Grosvenor square. C. 1889–90. *Sci. Com.* 1889—. *Referee*, 1884-8. *Trans.* 6.

Elected

1890 Ord, William Wallis, M.D., 32, Harley street, Cavendish square.

1877 Ormerod, Joseph Arderne, M.D., Physician to the National Hospital for the Paralysed and Epileptic, Queen square, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 25, Upper Wimpole street. *Trans.* 1.

1885 Ormsby, L. Hepenstal, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.

1879 Owen, Edmund, M.B., Surgeon to, and Joint Lecturer on Surgery at St. Mary's Hospital; Senior Surgeon to the Hospital for Sick Children, Great Ormond street; 64, Great Cumberland place, Hyde park. *Trans.* 2.

1882 Owen, Herbert Isambard, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 40, Curzon street, May Fair.

1874 Page, Herbert William, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. C. 1890-91. *Referee*, 1884-89. *Lib. Com.* 1886-8. *Trans.* 4.

1887 Paget, Charles Edward, North Bentcliffe, Eccles, Lancashire.

1840 †Paget, Sir James, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; Foreign Associate of the 'Académie de Médecine,' Paris; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1886 Paget, Stephen, 57, Wimpole street, Cavendish square.

Elected

1858 *Paley, William, M.D., Physician to the Ripon Dispensary; The Old Residence, Ripon, Yorkshire.

1887 Pardington, George Lucas, M.D., 47, Mount Pleasant road, Tunbridge Wells.

1873 Parker, Robert William, Senior Surgeon to the East London Hospital for Children; Surgeon to the German Hospital; 8, Old Cavendish street. C. 1888-9. *Lib. Com.* 1885-91. *Trans.* 4.

1885 Parker, Rushton, M.B., B.S., Professor of Surgery, University College, Liverpool (Victoria University); Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.

1891 Parkin, Alfred, M.S., M.D., 149, Beverley road, Hull. *Trans.* 1.

1889 Parsons, J. Inglis, M.D., 3, Queen street, May Fair.

1883 Pasteur, William, M.D., Assistant Physician to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 4 Chandos street, Cavendish square.

1891 Paterson, William Bromfield, 64, Brook street, Grosvenor square.

1865 Pavy, Frederick William, M.D., F.R.S., Consulting Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. *Referee*, 1871-82. *Trans.* 1.

1869 Payne, Joseph Frank, M.D., Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889-91.

1879 Peel, Robert, 120, Collins street east, Melbourne, Victoria.

1856 Peirce, Richard King, Laggan House, Maidenhead.

1830 Pelechin, Charles P., M.D., St. Petersburg.

1855 *Pemberton, Oliver, Senior Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 65, Temple row, Birmingham. *Trans.* 1.

Elected

1874 PENHALL, JOHN THOMAS, The Cedars, Broadwas-on-Thema, Worcester.

1887 PENROSE, FRANCIS GEORGE, M.D., Assistant Physician to St. George's Hospital; 4, Harley street, Cavendish square. *Sci. Com.* 1889—.

1890 PERRY, EDWIN COOPER, M.D., Assistant Physician and Demonstrator of Pathology at Guy's Hospital; The College, Guy's Hospital.

1879 *PESIKAKA, HORMASJI DOSABHAI, Marine Lines, Bombay.

1878 *PHILIPSON, GEORGE HARE, M.D., M.A., D.C.L., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., F.R.S.Ed., 10, Henrietta street, Cavendish square, W.

1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Leinster square, Bayswater.

1888 PHILLIPS, JOHN, M.A., M.D., Assistant Obstetric Physician, King's College Hospital; Physician to the British Lying-in Hospital; 71, Grosvenor street, Grosvenor square.

1889 PHILLIPS, SIDNEY, M.D., Senior Physician to Out-patients and Lecturer on Materia Medica at St. Mary's Hospital, Physician to the London Fever Hospital, and to the Lock Hospital; 62, Upper Berkeley street, Portman square.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. *Referee,* 1882-3. *Sci. Com.* 1870. *Lib. Com.* 1879-81.

1841 †PITMAN, SIR HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital; Cranbrook, Bycullah park, Enfield. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee,* 1849-50. *Lib. Com.* 1847.

1884 PITT, GEORGE NEWTON, M.D., Assistant Physician to, and Pathologist at, Guy's Hospital; 24, St. Thomas's street, Southwark. *Trans.* 1.

Elected

1889 PITTS, BERNARD, M.B., M.C., Assistant Surgeon to St. Thomas's Hospital; 31, Harley street, Cavendish square.

1885 POLAND, JOHN, Surgeon to the Miller Hospital, Greenwich; 4, St. Thomas's street, Southwark.

1884 POLLARD, BILTON, Assistant Surgeon to University College Hospital, Surgeon to the North-Eastern Hospital for Children; 24, Harley street, Cavendish square. *Trans.* 1.

1845 †POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. P. 1886-7. *Referee*, 1858, 1864-9, 1877-85. *Trans.* 5.

1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street. C. 1890-91. *Referee*, 1887-89. *Trans.* 2.

1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.

1846 POTTER, JEPHSON, M.D., F.L.S.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., Physician Extraordinary to H.M. the Queen; Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; 62, Wimpole street, Cavendish square. S. (Oct.), 1883-5. C. 1887-8. *Referee* 1879-83, 1886. *Trans.* 3.

1887 POWER, D'ARCY, M.A., M.B., Demonstrator of Practical Surgery at St. Bartholomew's Hospital; Surgeon to Out-patients at Victoria Hospital for Children; 26, Bloomsbury square.

Elected

1867 Power, Henry, Senior Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital, 37A, Great Cumberland place, Hyde Park. C. 1882-3. *Referee*, 1870-81, 1891—. *Sci. Com.* 1870. *Lib. Com.* 1872-8.

1857 †Priestley, William Overend, M.D., LL.D., Consulting Physician to King's College Hospital, and to the St Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.

1883 Pringle, John James, M.B., C.M., Assistant Physician to, and Physician in Charge of Skin Department at, the Middlesex Hospital, and Physician to the Royal Hospital for Diseases of the Chest; 23, Lower Seymour street, Portman square. *Trans.* 1.

1874 Purves, William Laidlaw, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

1878 Pye, Walter.

1877 Pye-Smith, Philip Henry, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 54, Harley street, Cavendish square. *Lib. Com.* 1887-91. *Trans.* 1.

1850 †Quain, Sir Richard, Bart., M.D., LL.D.Ed., F.R.S., Physician Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

1871 Ralfe, Charles Henry, M.D., M.A., Assistant Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square. C. 1889. *Referee*, 1885-8.

1857 Ranke, Henry, M.D., 3, Sophienstrasse, Munich.

Elected

1890 Ransom, William Bramwell, M.D., Physician to the Nottingham General Hospital; The Pavement, Nottingham.

1854 Ransom, William Henry, M.D., F.R.S., Consulting Physician to the Nottingham General Hospital; The Pavement, Nottingham.

1869 Read, Thomas Laurence, 11, Petersham terrace, Queen's gate.

1891 Reece, Richard James, 34, Eardley crescent, South Kensington.

1858 †Reed, Frederick George, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1882 Reid, James, M.D., C.B., Resident Physician in Ordinary to H.M. the Queen, Windsor Castle.

1884 Reid, Thomas Whitehead, Surgeon to the Kent and Canterbury Hospital; St. George's House, Canterbury, Kent.

1891 Remfry, Leonard, M.D., 4, Harley street, Cavendish square.

1855 †Reynolds, John Russell, M.D., F.R.S., Physician-in-Ordinary to H.M.'s Household; Consulting-Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.

1865 Rhodes, George Winter, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

1881 Rice, George, M.B., C.M., Sutton, Surrey.

1887 Richardson, Gilbert, M.D., Hawthorn House, Putney.

1863 Ringer, Sydney, M.D., F.R.S., Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80, 1889-91. *Trans.* 6.

1889 Rivers, W. H. Rivers, M.D., National Hospital, Queen Square.

1871 Rivington, Walter, M.S., Consulting Surgeon to the London Hospital; 95, Wimpole street, Cavendish square. C. 1885-6. *Trans.* 4.

Elected

1871 *Roberts, David Lloyd, M.D., Obstetric Physician to the Manchester Royal Infirmary, Physician to St. Mary's Hospital, Manchester; 11, St. John street, Manchester.

1878 Roberts, Frederick Thomas, M.D., Professor of Materia Medica and Therapeutics in University College, London; and Physician to University College Hospital; Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square. *Sci. Com.* 1889—.

1889 Roberts, Hugh Leslie, M.B., C.M., 31, Rodney street, Liverpool.

1889 Roberts, Sir William, M.D., B.A., F.R.S., 8, Manchester square. *Trans.* 2.

1857 Robertson, John Charles George, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 Robertson, William Henry, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital; Buxton, Derbyshire.

1888 *Robinson, Frederick William, M.B., C.M., Huddersfield.

1889 Robson, Arthur William Mayo, Hillary place, Leeds. *Trans.* 1.

1885 Rockwood, William Gabriel, M.D., Colombo, Ceylon.

1890 Rolleston, Humphry Davy, M.B., B.C., 13, Upper Wimpole street, Cavendish square.

1850 Roper, George, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; and to the Royal Infirmary for Children and Women, Waterloo Bridge road; Oulton Lodge, Aylsham, Norfolk. C. 1879-80.

1857 †Rose, Henry Cooper, M.D., F.L.S., Consulting Surgeon to the Hampstead Dispensary; 53, Rosslyn hill, Hampstead. C. 1886-7. *Trans.* 1.

1883 Rose, William, M.B., Professor of Surgery at King's College, Surgeon to King's College Hospital and to the Royal Free Hospital; 17, Harley street, Cavendish square.

Elected

1889 Ross, Daniel McClure, 54, Upper Berkeley street, Portman square.

1888 Roughton, Edmund Wilkinson, M.B., B.S., 33, Westbourne terrace, Hyde Park. *Trans.* 1.

1882 Routh, Amand Jules McConnel, M.D., B.S., Physician to the Samaritan Free Hospital for Women; Assistant Obstetric Physician to the Charing Cross Hospital; 14A, Manchester square.

1849 †Routh, Charles Henry Felix, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

1863 Rowe, Thomas Smith, M.D., Senior Visiting Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1882 Roy, Charles Smart, M.D., F.R.S., Professor of Pathology in the University of Cambridge; Trinity College, Cambridge.

1871 Rutherford, William, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1886 Sainsbury, Harrington, M.D., Physician to the Royal Free Hospital and Assistant Physician to the City of London Hospital for Diseases of the Chest; 63, Welbeck street, Cavendish square. *Trans.* 1.

1856 Salter, S. James A., M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

1855 †Sanderson, John Burdon, M.D., LL.D., D.C.L. Durham, F.R.S., Waynflete Professor of Physiology in the University of Oxford; 50, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

1867 Sandford, Folliott James, M.D., Market Drayton, Shropshire.

Elected

1879 Sangster, Alfred, B.A., M.B., Physician to the Skin Department, and Demonstrator of Skin Diseases at the Charing Cross Hospital; 6, Savile row. *Trans.* 1.

1869 Sansom, Arthur Ernest, M.D., Senior Physician to the North-Eastern Hospital for Children; Physician (with charge of out-patients) to the London Hospital; 84, Harley street, Cavendish square. C. 1887-8. *Referee*, 1889-91. *Trans.* 2.

1891 Santi, Philip Robert William, 32, St. Mary's place, Newcastle.

1886 Saundby, Robert, M.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; 83a, Edmund street, Birmingham.

1845 †Saunders, Sir Edwin, Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales; 13a, George street, Hanover square. C. 1872-3.

1891 Saunders, Frederick William, M.B., B.C., 17, Barkston gardens, South Kensington.

1834 Sauvan, Ludwig V., M.D., Warsaw.

1879 Savage, George Henry, M.D., Lecturer on Mental Diseases at Guy's Hospital; 3, Henrietta street, Cavendish square.

1859 Savory, Sir William Scovell, Bart., F.R.S., Surgeon Extraordinary to H.M. the Queen, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. V.P. 1883-4. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 8.

1883 Schäfer, Edward Albert, F.R.S., Jodrell Professor of Physiology, University College, London; University College, Gower street. *Referee*, 1888-91. *Sci. Com.* 1889-91.

1887 Scott, Harry, M.D., 47, St. Ermin's mansions, Westminster.

Elected

1861 *Scott, William, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1882 Scriven, John Barclay, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

1863 Sedgwick, William, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.

1877 Semon, Felix, M.D., Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole street, Cavendish square. *Trans.* 1.

1882 Sharkey, Seymour John, M.D., Physician and Joint Lecturer on Pathology at St. Thomas's Hospital; 2, Portland place. *Trans.* 2.

1840 Sharp, William, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1886 Shaw, Lauriston Elgie, M.D., Assistant Physician to Guy's Hospital; 10, St. Thomas's street, Southwark.

1884 Sheild, Arthur Marmaduke, M.B., B.S., Assistant Surgeon, Charing Cross Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

1890 Sheppard, Charles Edward, M.D. (deceased).

1859 †Sibley, Septimus William, 7, Harley street, Cavendish square. C. 1882-3. *Sci. Com.* 1863. *Trans.* 4.

1887 Sidebotham, Edward John, M.B., Erlesdene, Bowdon, Cheshire.

1848 †Sieveking, Sir Edward Henry, M.D., LL.D., Physician-in-Ordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. P. 1888-9. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

Elected

1886 Silcock, Arthur Quarry, M.D., B.S., Surgeon in charge of out-patients, St. Mary's Hospital; Assistant Surgeon, Royal London Ophthalmic Hospital; 52, Harley street, Cavendish square.

1842 †Simon, Sir John, K.C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

1857 Siordet, James Lewis, M.B., Villa Preti, Mentone, Alpes Maritimes, France.

1890 Smale, Morton, 22a, Cavendish square.

1879 Smith, E. Noble, Senior Surgeon and Surgeon to the Orthopædic Department of the Farringdon Dispensary; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881 Smith, Eustace, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 15, Queen Anne street, Cavendish square.

1866 Smith, Heywood, M.A. M.D., 18, Harley street, Cavendish square.

1886 Smith, Howard Lyon.

1885 Smith, James Greig, M.B., C.M., F.R.S.Ed., Surgeon to the Bristol Royal Infirmary; 16, Victoria square, Clifton, Bristol.

1872 Smith, T. Gilbart, M.A., M.D., Assistant-Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. C. 1890. *Trans.* 1.

1889 Smith, Robert Percy, M.D., B.S., Resident Physician and Medical Superintendent, Bethlem Royal Hospital, St. George's road, Lambeth.

1838 †Smith, Spencer, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

Elected

1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.

1874 *SMITH, WILLIAM ROBERT, M.D., D.Sc., F.R.S.Ed., Professor of Forensic Medicine at King's College, London; 74, Great Russell Street, Bloomsbury. *Trans.* 1.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.

1844 SPACKMAN, FREDERICK ROBERT, M.D., Consulting Physician to St. Alban's Hospital, Harpenden, St. Alban's.

1889 SPENCER, HERBERT R., M.D., B.S., 10, Mansfield street, Portland place.

1887 SPENCER, WALTER GEORGE, M.B., Assistant Surgeon to the Westminster Hospital; 35, Brook street, Grosvenor square. *Trans.* 1.

1888 SPICER, ROBERT HENRY SCANES, M.D., Physician to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck street, Cavendish square.

1890 SPICER, WILLIAM THOMAS HOLMES, M.B., 6A, Bedford square.

1875 SPITTA, EDMUND JOHNSON, Ivy House, Clapham Common, Surrey.

1851 †SPITTA, ROBERT JOHN, M.D., East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1885 SQUIRE, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption; 53, Harley street, Cavendish square. *Trans.* 1.

Elected

1854 Stevens, Henry, M.D., Inspector, Medical Department, Local Government Board, Whitehall; Falcon Lodge, Hampton, Middlesex.

1884 Stewart, Edward, M.D., 8, Upper Wimpole street, Cavendish square.

1859 †Stewart, William Edward, 16, Harley street, Cavendish square.

1879 *Stirling, Edward Charles, Adelaide, South Australia [care of Messrs. Elder and Co., 7, St. Helen's place].

1856 †Stocker, Alonzo Henry, M.D., Peckham House, Peckham.

1865 Stokes, Sir William, M.D., M.C., Surgeon to the Meath Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1884 Stonham, Charles, Assistant Surgeon to the Westminster Hospital, and Curator of Anatomical Museum, University College, London; 4, Harley street, Cavendish square.

1843 Storks, Robert Reeve.

1871 Strong, Henry John, M.D., Surgeon to the Croydon General Hospital; Whitgift House, George street, Croydon.

1863 †Sturges, Octavius, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9. V.P. 1889. *Referee*, 1882-8.

1871 †Sutherland, Henry, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

1883 Sutton, John Bland, Assistant Surgeon, Lecturer on Comparative Anatomy, and Senior Demonstrator of Anatomy to the Middlesex Hospital; 48, Queen Anne street, Cavendish square. *Trans.* 5.

1890 Syers, Henry Walter, M.D., 3, Devonshire street, Portland place.

Elected

1886 SYMONDS, CHARTERS JAMES, M.S., Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; 26, Weymouth street, Portland place.

1890 SYMPSON, E. MANSEL, M.A., M.B., B.C., 5, James street, Lincoln.

1878 *SYMPSON, THOMAS, Surgeon to the Lincoln County Hospital; 3, James street, Lincoln.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 4.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1875 TAY, WAREN, Surgeon to the London Hospital, to the Royal London Ophthalmic Hospital, to the North Eastern Hospital for Children, and to the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., *Secretary;* Physician to, and Lecturer on Medicine at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 20, Wimpole street, Cavendish square. S. 1889-91. *Sci. Com.* 1889—. *Referee,* 1887-8. *Trans.* 2.

1890 TAYLOR, SEYMOUR, M.D., Assistant Physician West London Hospital; 16, Seymour street, Portman square.

1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's Wood.

1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's. C. 1888-9.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 10.

1890 THOMAS, WILLIAM ROBERT, M.D., Forest House, Bath road, Bournemouth.

1862 THOMPSON, EDMUND SYMES, M.D., Senior Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Sci. Com.* 1889—. *Referee,* 1876-7. *Trans.* 1.

Elected

1852 †Thompson, Sir Henry, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Member of the "Société de Chirurgie," Paris; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 8.

1862 Thompson, Reginald Edward, M.D., Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1881 Thomson, William Sinclair, M.D., late Senior Consulting Surgeon to Peterbro' Hospital, and Medical Officer of Health for Peterbro'; 1, Palace court, Notting Hill gate.

1876 Thornton, John Knowsley, M.B., C.M., Consulting Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman street, Portman square. C. 1891. *Lib. Com.* 1886-90. *Trans.* 5.

1883 Thursfield, Thomas William, M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.

1848 †Tilt, Edward John, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square. *Referee*, 1874-81.

1889 Tirard, Nestor Isidore Charles, M.D., Professor of Materia Medica and Therapeutics, King's College; Senior Assistant Physician to King's College Hospital, and Physician to the Evelina Hospital for Sick Children; 28, Weymouth street, Portland place.

1880 Tivy, William James, 8, Lansdowne place, Clifton, Bristol.

1872 Tomes, Charles Sissmore, M.A., F.R.S., 37, Cavendish square. C. 1887. *Lib. Com.* 1879.

1882 Tooth, Howard Henry, M.D., Assistant Medical Tutor St. Bartholomew's Hospital; 34, Harley street, Cavendish square.

1871 *Trend, Theophilus W., M.D., 1, Grosvenor square, Southampton.

Elected

1879 TREVES, FREDERICK, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 6, Wimpole street, Cavendish square. *Referee*, 1890-91. *Sci. Com.* 1889-91. *Trans.* 5.

1881 *TREVES, WILLIAM KNIGHT, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate. *Sci. Com.* 1889—.

1867 TROTTER, JOHN WILLIAM, late Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1889 TURNBULL, GEORGE LINDSAY, M.B., 121, Ladbroke grove.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the North-Eastern Hospital for Children, and to the London Hospital; 15, Finsbury square.

1873 TURNER, GEORGE BROWN, M.D., Vernon House, Ryde, Isle of Wight.

1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Assistant Surgeon to, and Joint Lecturer on Practical Surgery at, St. George's Hospital; 49, Green street, Park lane.

1891 TWEED, REGINALD, M.D., 55, Upper Brook street, Grosvenor square.

1888 TYLDEN, HENRY JOHN, M.B., 38, Harewood square.

1881 TYSON, WILLIAM JOSEPH, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne Gardens, Folkestone.

1876 VENN, ALBERT JOHN, M.D., Obstetric Physician to the Metropolitan Free Hospital; Physician for the Diseases of Women, West London Hospital; 122, Harley street, Cavendish square.

1870 VENNING, EDGCOMBE, 30, Cadogan place.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.

Elected

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy, and to the French Hospital, Leicester place; 19A, Hanover square.

1891 VOELCKER, ARTHUR FRANCIS, M.D., B.S., Pathologist and Curator of the Museum at the Middlesex Hospital; 13, Welbeck street, Cavendish square.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

1886 WAINEWRIGHT, BENJAMIN, M.B., C.M., 67, Grosvenor street, Grosvenor square.

1864 WAITE, CHARLES DERBY, M.B., Consulting Physician to the Westminster General Dispensary; 3, Old Burlington street.

1884 WAKLEY, Thomas, jun., 5, Queen's Gate, South Kensington.

1868 *WALKER, ROBERT, Honorary Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.

1887 WALLACE, EDWARD JAMES, M.D., Holmbush, Grove road, Southsea.

1883 WALLER, AUGUSTUS, M.D., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road.

1888 WALLIS, FREDERICK CHARLES, M.B., B.C., 18, St. James's street.

1867 WALLIS, GEORGE, Surgeon to Addenbrooke's Hospital, Corpus Buildings, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Assistant Surgeon to, and Demonstrator of Practical and Orthopædic Surgery at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 27, Weymouth street, Portland place. C. 1888-9. *Lib. Com.* 1882-5. *Trans.* 5.

1852 †WALSHE, WALTER HAYLE, M.D., LL.D.Edin., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption and to University College Hospital; 41, Hyde Park square. C. 1872. *Trans.* 1.

Elected

1883 *Walters, James Hopkins, Surgeon to the Royal Berkshire Hospital; 15, Friar street, Reading.

1886 Ward, Allan Ogier, M.D., Lansdowne House, High road, Tottenham.

1890 Ward, Arthur Henry, 7, Hertford street, May Fair.

1821 Ward, William Tilleard, Tilleards, Stanhope, Canada.

1846 Ware, James Thomas, Tilford House, near Farnham, Surrey.

1891 Waring, Holburt Jacob, St. Bartholomew's Hospital.

1877 Warner, Francis, M.D., Assistant Physician and Lecturer on Botany to the London Hospital; 5, Prince of Wales Terrace, Kensington Palace. *Trans.* 1.

1889 Washbourn, John Wychenford, M.D., Assistant Physician and Demonstrator of Bacteriology at Guy's Hospital; Guy's Hospital.

1861 Waters, A. T. Houghton, M.D., Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.

1861 †Watson, William Spencer, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.

1879 de Watteville, Armand, M.A., M.D., B.Sc., Physician in Charge of the Electro-therapeutical Department at St. Mary's Hospital; 30, Welbeck street, Cavendish square.

1840 Webb, William Woodham, M.D., Neuilly-sur-Seine, France.

1891 Weber, Frederic Parkes, M.B., St. Bartholomew's Hospital.

1857 Weber, Hermann, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Sci. Com.* 1889—. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.

1844 †Wegg, William, M.D., 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-80. *Lib. Com.* 1851-3.

Elected

1878 Weiss, Hubert Foveaux, Assistant Surgeon to the West London Hospital; 6, Granville gardens, Ramsgate, and [27, Piccadilly].

1874 Wells, Harry, M.D., San Ysidro, Buenos Ayres, S. America.

1854 †Wells, Sir Thomas Spencer, Bart., Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Corresponding Member, "Académie de Médecine," Paris; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 14. *Pro.* 1.

1842 †West, Charles, M.D., Foreign Associate of the Academy of Medicine of Paris; Kenilworth, Eaton road, West Brighton. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 West, Samuel, M.D., Assistant Physician to St. Bartholomew's Hospital; Senior Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square. *Trans.* 4.

1888 Wethered, Frank Joseph, M.D., Assistant Physician City of London Hospital for Diseases of the Chest, Victoria Park; 34, Queen Anne street, Cavendish square. *Trans.* 1.

1882 Wharry, Charles John, M.D.

1881 Wharry, Robert, M.D., Physician to the Westminster Dispensary; 6, Gordon square.

1878 Wharton, Henry Thornton, M.A., Honorary Surgeon to the Kilburn Dispensary; "Madresfield," Acol road, Priory road, West Hampstead.

1828 Whatley, John, M.D.

1875 Whipham, Thomas Tillyer, M.B., Physician to, and Lecturer on Pathology and Practical Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square.

1849 White, John.

Elected

1881 WHITE, WILLIAM HALE, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Referee*, 1888-91. *Trans.* 3.

1890 WHITE-COOPER, G. O., M.B., 5, Cranley gardens, Brompton.

1881 *WHITEHEAD, WALTER, F.R.S. Ed., Senior Surgeon to the Manchester Royal Infirmary, and to the Manchester and Salford Lock and Skin Hospital; 499, Oxford road, Manchester. *Trans.* 1.

1885 *WHITLA, WILLIAM, M.D., Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.

1877 WHITMORE, WILLIAM TICKLE, Surgeon to the Westminster General Dispensary; 7, Arlington street, Piccadilly.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1870 *WILKIN, JOHN F., M.D., M.C., The Warren, Beckenham park, Kent.

1883 *WILKINSON, THOMAS MARSHALL, Surgeon to the Lincoln County Hospital and to the Lincoln General Dispensary; 33, Avenue road, Grantham.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., LL.D., F.R.S., Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught, and to H.R.H. the Duke of Edinburgh; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street, Grosvenor square. *Referee*, 1872-81. *Sci. Com.* 1.

1883 *WILLANS, WILLIAM BLUNDELL, Great Hadham, Herts.

1890 WILLCOCKS, FREDERICK, M.D., Assistant Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Physician to Out-Patients at the Evelina Hospital for Sick Children; 14, Mandeville street, Manchester square.

Elected

1865 †Willett, Alfred, Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. V.P. 1890-91. *Referee,* 1882-89. *Trans.* 2.

1887 Willett, Edgar William, M.B., 25, Welbeck street, Cavendish square.

1864 Willett, Edmund Sparshall, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1888 Williams, Campbell.

1859 *Williams, Charles, Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.

1866 Williams, Charles Theodore, M.A., M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook street, Grosvenor square. C. 1884-5. *Referee,* 1888-91. *Lib. Com.* 1880-3. *Sci. Com.* 1889—. *Trans.* 5.

1881 Williams, Dawson, M.D., Assistant Physician to the East London Hospital for Children; 25, Old Burlington street.

1872 Williams, John, M.D., Physician Accoucheur to H.R.H. the Princess Beatrice; Professor of Midwifery, University College, London; Obstetric Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1891. *Referee,* 1878-90. *Lib. Com.* 1876-82.

1868 Williams, William Rhys, M.D., Linden House, Bertie road, Leamington.

1890 Wills, William Alfred, M.D., 52, Davies street, Berkeley square.

1887 Wilson, Arthur Hervey, M.D., 504, Broadway, Boston, U.S.A.

1889 Wilson, John Henry Parker, H.M.'s Military Prison, The Avenue, Brixton Hill.

1863 Wilson, Robert James, 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1889 Wise, A. Tucker, M.D., Kursaal de la Maloja.

Elected

1850 *Wise, Robert Stanton, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn-Banbury.

1879 Woakes, Edward, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.

1885 Wolfenden, Richard Norris, M.D., Assistant Physician to the North-West London Hospital; 35, Harley street, Cavendish square.

1851 †Wood, John, F.R.S., 61, Wimpole street, Cavendish square. C. 1867-8. V.P. 1877-8. *Referee*, 1871-6, 1880-89. *Lib. Com.* 1866. *Trans.* 3.

1887 Wood, Thomas Outterson, M.D., 40, Margaret street, Cavendish square.

1848 †Wood, William, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8. V.P. 1877-8.

1883 Wood, William Edward Ramsden, M.A., M.D., The Priory, Roehampton.

1891 Woodforde, Alfred Pownall, 160, Goldhawk road, W.

1879 Woodward, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1890 Wynter, Walter Essex, M.D., B.S., Medical Registrar, Middlesex Hospital; 30, Upper Berkeley street, Portman square.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Hon. Secretaries before the 1st of September in each year, in order that the List may be made as correct as possible.]

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION.

1838 Charles Hawkins.
Henry Spencer Smith.
1840 Sir James Paget, Bt., F.R.S.
1841 Sir William Bowman, Bart., F.R.S.
Paul Jackson.
1842 Sir John Simon, K.C.B., F.R.S.
John Erichsen F.R.S.
Sir Oscar M. P. Clayton, C.M.G.
1843 Henry Lee.
Edward Newton.
1844 William Wegg, M.D.
1845 George D. Pollock.
Thomas Taylor.
Sir Edwin Saunders.
Edward U. Berry.
1846 John A. Bostock.
Barnard Wight Holt.
1847 George Johnson, M.D., F.R.S.
1848 Sir Edward H. Sieveking, M.D.
William Wood, M.D.
Thomas Hawksley, M.D.
Edward John Tilt, M.D.
1849 C. H. F. Routh, M.D.
1850 Sir Richard Quain, Bt., M.D., F.R.S
1851 John Birkett.
John A. Kingdon.
Peter Y. Gowlland.
John Wood, F.R.S.
Bernard E. Brodhurst.
Robert J. Spitta, M.D.
1852 Walter H. Walshe, M.D.
William Adams.
Sir Henry Thompson.
1853 Robert Brudenell Carter.
1854 Sir Alfred Baring Garrod, M.D., F.R.S.
Sir Thomas Spencer Wells, Bt.
1855 W. M. Graily Hewitt, M.D.
J. Burdon Sanderson, M.D., F.R.S.
J. Russell Reynolds, M.D., F.R.S.
William Marcet, M.D., F.R.S.
1856 Charles J. Hare, M.D.
William Bird.
Jonathan Hutchinson, F.R.S.
Timothy Holmes.
Alonzo H. Stocker, M.D.
1857 William Overend Priestley, M.D.
George Harley, M.D., F.R.S.
Hermann Weber, M.D.
John Whitaker Hulke, F.R.S.
John Morgan.
Henry Cooper Rose, M.D.
Henry Walter Kiallmark.
1858 Fred. George Reed, M.D.
John William Ogle, M.D.
1859 Wm. Howship Dickinson, M.D.
Sir William Scovell Savory, Bart., F.R.S.
Edwin Thomas Truman.
Richard Barwell.
Edward Tegart.
Septimus William Sibley.
William E. Stewart.
1860 Sir Andrew Clark, Bt., M.D., F.R.S.
William Ogle, M.D.
Thomas Bryant.
John Couper.

1860 Henry Howard Hayward.
1861 Robert Barnes, M.D.
William Spencer Watson.
1862 James Andrew, M.D.
Lionel Smith Beale, M.B., F.R.S.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D.
William Henry Brace, M.D.
George Cowell.
Robert Farquharson, M.D., M.P.
M. Berkeley Hill.
1863 Octavius Sturges, M.D.
John Langdon H. Down, M.D.
Samuel Wilks, M.D., F.R.S.
Samuel Fenwick, M.D.
Julius Althaus, M.D.
Sydney Ringer, M.D., F.R.S.
Thomas Smith.
Arthur B. R. Myers.
Arthur E. Durham.
William Sedgwick.
1864 George Buchanan, M.D., F.R.S.
Charles Derby Waite, M.B.
John Harley M.D.
Thomas William Nunn.
1865 Charles Robert Drysdale, M.D.
James Edward Pollock, M.D.
William Cholmeley, M.D.
Reginald Southey, M.D.
George Fielding Blandford, M.D.
Sir Dyce Duckworth, M.D.
Frederick W. Pavy, M.D., F.R.S.
William Morrant Baker.
John Langton.
Frederick James Gant.
Alfred Willett.
Bowater John Vernon.
Alfred Cooper.
Christopher Heath.
1866 Thomas Fitz-Patrick, M.D.
Samuel Jones Gee, M.D.
Charles Theodore Williams, M.D.
Heywood Smith, M.D.
William Selby Church, M.D.
Thomas Clifford Allbutt, M.D., F.R.S.
1867 William Henry Day, M.D.
Achille Vintras, M.D.
Richard Douglas Powell, M.D.
F. Howard Marsh.
Henry Power.
Sir William MacCormac.
Thomas Pickering Pick.
Charles Arthur Aikin.
1868 H. Charlton Bastian, M.D., F.R.S.
William Henry Broadbent, M.D.
Thomas Buzzard, M.D.
John Cavafy, M.D.
Walter Butler Cheadle, M.D.
John Cockle, M.D.
Sir Thos. Crawford, K.C.B., M.D.
T. Henry Green, M.D.
William Chapman Grigg, M.D.
John Croft.
George Eastes.
William Henry Freeman.
1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.
Thomas Laurence Read.
1870 J. Warrington Haward.
Edgcombe Venning.
Clement Godson, M.D.
Reginald Harrison.
1871 William Cayley, M.D.
Charles Henry Ralfe, M.D.
Thomas L. Brunton, M.D., F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
Henry Sutherland, M.D.
George Vivian Poore, M.D.
Walter Rivington.
Marcus Beck.
Benjamin Duke.
1872 Gilbart Smith, M.D.
Thomas B. Christie, M.D.
George B. Brodie, M.D.
John Williams, M.D.
Sir J. Fayrer, M.D., F.R.S.
Charles S. Tomes, B.A., F.R.S.
Sir William Bartlett Dalby.
1873 William Miller Ord, M.D.
Frederick Taylor, M.D.
Norman Moore, M.D.
John Curnow, M.D.
William R. Gowers, M.D., F.R.S.
Sir Wm. Guyer Hunter, M.D., M.P.
Jeremiah McCarthy.
Wm. Johnson Smith.
Robert William Parker.
Alex. O. McKellar.
Henry T. Butlin.
Charles Higgens.
William J. Walsham.
Edward Milner.
1874 Alfred Lewis Galabin, M.D.
George Thin, M.D.
Alfred B. Duffin, M.D.

1874 James H. Aveling, M.D.
John Mitchell Bruce, M.D.
Henry Morris.
William Laidlaw Purves.
William Harrison Cripps.
Henry G. Howse.
Herbert William Page.
Frederic Durham.
John J. Merriman.
William Robert Smith, M.D.
1875 Thomas T. Whipham, M.B.
Francis Charlewood Turner, M.D.
Thomas Crawford Hayes, M.D.
Charles Henry Carter, M.D
Fletcher Beach, M.B
Waren Tay.
Edmund J. Spitta.
1876 Thomas Barlow, M.D.
Wm. Lewis Dudley, M.D.
Albert J. Venn, M.D.
John Knowsley Thornton.
Charles Macnamara.
John N. C. Davies-Colley.
1877 Felix Semon, M.D.
Sidney Coupland, M.D.
Francis Warner, M.D.
William Ewart, M.D.
Alfred Pearce Gould.
Rickman J. Godlee.
Alban H. G. Doran.
George Ernest Herman, M.B.
Samuel West, M.D.
John Abercrombie, M.D.
George Allan Heron, M.D.
Joseph A. Ormerod, M.D.
P. Henry Pye-Smith, M.D., F.R.S.
Edward Nettleship.
William Henry Bennett.
William T. Whitmore.
1878 Sir Jas. Crichton Browne, M.D.
Fred. T. Roberts, M.D.
Sir Joseph Lister, Bart., F.R.S.
Clinton T. Dent.
John H. Morgan.
Donald W. Charles Hood, M.B.
Henry Gervis, M.D.
Richard Davy.
Hubert Foveaux Weiss.
Henry Thornton Wharton.
1879 Alfred Sangster, M.B.
Edward Woakes, M.D.
Armand de Watteville, M.D.
Malcolm A. Morris.
A. E. Cumberbatch.
1879 Edmund Owen.
Arthur E. J. Barker.
Frederick Treves.
Horatio Donkin, M.B.
Thomas John Maclagan, M.D.
David White Finlay, M.D.
Andrew Clark.
Francis Henry Champneys, M.B.
William Watson Cheyne.
William Munk, M.D.
George Henry Savage, M.D.
H. H. Clutton, M.A.
Frederic S. Eve.
E. Noble Smith.
William Henry Allchin, M.B.
F. G. Dawtrey Drewitt, M.D.
1880 Robert Alex. Gibbons, M.D.
David Ferrier, M.D., F.R.S.
Vincent Dormer Harris, M.D.
Edmund Distin Maddick.
Jas. John MacWhirter Dunbar, M.B.
James William Browne, M.B.
William Appleton Meredith, M.B.
Alexander Hughes Bennett, M.D.
Malcolm Macdonald McHardy.
Alexander Wm. Macfarlane, M.D.
A. Boyce Barrow.
William Murrell, M.D.
Leslie Ogilvie, M.B.
George Ogilvie, M.B.
Charles Edward Beevor, M.D.
Thomas Colcott Fox, M.B.
George Henry Makins.
1881 Francis de Havilland Hall, M.D.
Robert Wharry, M.D.
Cecil Yates Biss, M.D.
Richard Clement Lucas.
Stephen Mackenzie, M.D.
James Anderson, M.D.
William Hale White, M.D.
Eustace Smith, M.D.
William Sinclair Thomson, M.D.
Percy Kidd, M.D.
Oswald A. Browne, M.A.
W. Bruce Clarke, M.B.
Dawson Williams, M.D.
George Lindsay Johnson, M.A., M.D.
Henry Edward Juler.
Jonathan F. C. H. Macready.
C. B. Lockwood.
1882 Philip J. Hensley, M.D.
Ernest Clarke, M.D.
John Barclay Scriven.

1882 George Robertson Turner.
Howard Henry Tooth, M.D.
Herbert Isambard Owen, M.D.
Charles R. B. Keetley.
Joseph Mills.
A. T. Myers, M.D.
Anthony A. Bowlby.
Amand J. McC. Routh, M.D.
Seymour J. Sharkey, M.D.
William Lang.
Henry Radcliffe Crocker, M.D.

1883 Edwin Clifford Beale, M.A., M.B.
James Kingston Fowler, M.D.
James Frederic Goodhart, M.D.
John Charles Galton, M.A.
Walter Hamilton Acland Jacobson.
Walter H. Jessop, M.B.
Walter Edmunds, M.C.
Victor A. Horsley, F.R.S.
Dudley Wilmot Buxton, M.D.
Charles Douglas F. Phillips, M.D.
Angel Money, M.D.
John James Pringle, M.B.
Henry Roxburgh Fuller, M.D.
Wilmot Parker Herringham, M.D.
Augustus Waller, M.D.
William Pasteur, M.D.
Edward Albert Schäfer, F.R.S.
John Bland Sutton.
William Rose, M.B.
Storer Bennett.
Robert Marcus Gunn, M.B.
James Dixon Bradshaw, M.B.
George Knapton.

1884 George Newton Pitt, M.D.
Charles Stonham.
Stanley Boyd, M.B.
William Arbuthnot Lane, M.S.
Dennis Dallaway.
Thomas Whitehead Reid.
Arthur Marmaduke Sheild, M.B.
Frederic Bowreman Jessett.
Sidney Harris Cox Martin, M.B.
George Lawson.
Thomas Wakley, Jun.
Robert James Lee, M.D.
F. Swinford Edwards.
James Johnston, M.D.
Edward Stewart, M.D.
William Duncan, M.D.
Charles Chinner Fuller.
Jean Samuel Keser, M.D.
George Richard Turner Phillips.
Bilton Pollard.

1885 Alexander Haig, M.B.
Wm. Dobinson Halliburton, M.D.
Theodore Dyke Acland, M.D.
Frederick Walker Mott, M.D.
William Maunsell Collins, M.D.
James Berry.
John Cahill.
Francis Henry Hawkins, M.B.
John Poland.
Heinrich Port, M.D.
Edward Emanuel Klein, M.D., F.R.S.
R. Norris Wolfenden, M.D.
A. C. Butler-Smythe.
Charles Alfred Ballance, M.S.
Walter Spencer Anderson Griffith, M.B.
John Edward Squire, M.D.
John D. Malcolm, M.B., C.M.
Phineas S. Abraham, M.D.
Henry Willingham Gell, M.B.

1886 Robert Maguire, M.D.
Harrington Sainsbury, M.D.
Cuthbert Hilton Golding-Bird, M.S.
Benjamin Wainewright, M.B., C.M.
Charles Elliott Leopold Barton Hudson.
Lauriston Elgie Shaw, M.D.
Charters James Symonds, M.S.
Robert Boxall, M.D.
Allan Ogier Ward, M.D.
Archibald Edward Garrod, M.D.
Stephen Paget.
William Radford Dakin, M.D.
Samuel Herbert Habershon, M.D.
Arthur Quarry Silcock.
Arthur Hamilton Nicholson Lewers, M.D.

1887 Walter George Spencer.
Thomas Outterson Wood, M.D.
Richard Hingston Fox, M.D.
Edgar William Willett, M.B.
Henry Lewis Jones, M.D.
Francis George Penrose, M.D.
Hugh Percy Dunn.
Charles Edward Paget.
Frederic William Hewitt, M.D.
Harry Scott, M.D.
James Barry Ball, M.D.
Gilbert Richardson, M.D.
Edward James Wallace, M.D.
D'Arcy Power, M.B.
John Gay.
James Calvert, M.D.

1887 Percy J. F. Lush, M.B.
1888 Robert Henry Scanes Spicer, M.D.
Jonathan Hutchinson, Jun.
Campbell Williams.
Walter Baugh Hadden, M.D.
James Donelan, M.B., C.M.
John Anderson, M.D., C.I.E.
Laurie Asher Lawrence.
Arthur Pearson Luff, M.B., B.Sc.
Albert Carless, M.B., B.S.
Henry John Tylden, M.B.
Frederick Charles Wallis, M.B., B.C.
Charles James Cullingworth, M.D.
Edmund Cautley, M.B., B.C.
H. Montague Murray, M.D.
Arthur Symons Eccles, M.B.
Frank Joseph Wethered, M.B.
Edmund Wilkinson Roughton, M.D.
Edward Dillon Mapother, M.D.
Frederick William Cook, M.D.
John Phillips, M.B.
George Lindsay Turnbull, M.B.
1889 Montagu Handfield-Jones, M.D.
Norman MacMillan MacLehose, M.B.
David Henry Goodsall.
Raymond Johnson, M.B.
John Fletcher Little, M.B.
Henry Work Dodd.
W. H. Rivers Rivers, M.D.
Sir William Roberts, M.D., F.R.S.
Sidney Phillips, M.D.
Ernest le Cronier Lancaster, M.B.
William Charles Bull, M.B.
George P. Field.
John Wychenford Washbourn, M.D.
John Henry Parker Wilson.
Arthur J. M. Bentley, M.D.
Charles Henry Cosens.
Henry Percy Dean, M.B., B.S.
Sheridan Delépine, B.S., M.B., C.M.
Alfred Samuel Gubb.
William Hunter, M.D.
J. Inglis Parsons, M.D.
Bernard Pitts, M.B., M.C.
Daniel McClure Ross.
Robert Percy Smith, M.D., B.S.
Herbert R. Spencer, M.D., B.S.
Nestor Isidore Charles Tirard, M.D.
1890 John Rose Bradford, M.B.
Roland Danvers Brinton, M.D.
1890 James Cagney, M.D.
Charles D. B. Hale, M.D.
Edwin Cooper Perry, M.D.
Morton Smale.
Frederick Willcocks, M.D.
R. Ashton Bostock.
Henry Cripps Lawrence.
William T. Holmes Spicer, M.B.
David Anderson Berry.
Thomas Henry Crowle.
William Bramwell Ransom, M.D.
Henry Walter Syers, M.D.
Seymour Taylor, M.D.
William Alfred Wills, M.D.
G. O. White-Cooper, M.B.
Herbert William Allingham.
William Anderson.
William A. F. Bateman.
James Jackson Clarke, M.B.
Charles E. H. Cotes, M.B.
Leonard G. Guthrie, M.B., B.S.
Albert T. Hickson, M.A., M.D.
G. William Hill, M.D., B.Sc.
Edward Law, M.D., C.M.
Patrick Manson, M.D., C.M.
William Wallis Ord, M.D.
Humphry D. Rolleston, M.B., B.C.
Charles E. Sheppard, M.D. (dec.).
Arthur Henry Ward.
1891 Walter Essex Wynter, M.D., B.S.
William Lee Dickinson, M.B.
Herbert P. Hawkins, M.B., B.C.
Cyril Ogle, M.A., M.B.
Leonard Remfry, M.D.
Frederick W. Saunders, M.B., B.C.
Arthur F. Voelcker, M.D., B.S.
Alfred Pownall Woodforde.
Charles Gordon Brodie.
Herbert George Cook, M.B.
George Elam.
Herbert T. Herring, M.B., B.S.
Ernest Muirhead Little.
Henry Charrington Martin, M.D.
Frederick William Andrewes, M.B.
Charles Ernest Baker, M.B.
Wilfred Balgarnie, M.B.
Alfred Eddowes, M.D.
Herbert Morley Fletcher, M.B.
William Heaton Hamer, M.B.
John Arthur Hayward, M.B.
William Bromfield Paterson.
Reginald Tweed, M.D.
Holburt Jacob Waring.
Frederic Parkes Weber, M.B.

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The *Proceedings* are issued after each Meeting.

They are sent, postage free, to every Fellow of the Society who, in writing, expresses a wish to receive them.

They may be had by others at the Society's House, on payment (in advance) of an annual subscription of five shillings and eightpence, which may be sent either by post-office order or in postage-stamps;—this will include the expense of conveyance by post to any place within the Postal Union. For places beyond the Postal Union special arrangements must be made.

A notice of every paper will appear in the *Proceedings*. Authors will be at liberty, on sending their communications, to intimate to the Secretaries whether they wish them to appear in the *Proceedings* only, or in the *Proceedings* and *Transactions;* and in all cases they must furnish an Abstract of the communication.

The Abstracts of the papers read are sent to the Journals.

ADVERTISEMENT.

The Council of the Royal Medical and Chirurgical Society deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its 'Transactions.'

ROYAL MEDICAL AND CHIRURGICAL SOCIETY.

REPORT OF THE BUILDING COMMITTEE,

As amended and adopted by the Council,
February 5th, 1891.

Gentlemen—

As the building operations are now nearly completed, and the new lease from the City can almost immediately be granted, the time is approaching when the security for the Debenture Loan may be completed and the Debentures issued.

The Building Committee therefore beg leave to lay before the Council the following report of their proceedings, and to suggest to the Council to call a meeting of the Society for the purpose of confirming their action and obtaining authority for raising the extra funds required to pay the outstanding debts of the Society for building and other expenses.

The first idea of acquiring the present premises arose about February, 1889, when it was found that the house in Hanover Square might be purchased from two gentlemen who had just acquired it as a building speculation. This idea was developed by the Council in a report issued to the Fellows of the Society on February 27th, 1889. A reference to that report will show that the scheme then proposed was much less ambitious than the one now completed. The expenditure was estimated at only £4,300 beyond the purchase-money (£23,000); no provision was made for any new building, except that of a meeting-room, at a cost of £1,500; no enlargement of the old house was contemplated, nor any new building on the leasehold ground. The details of estimated expenditure in that report were merely conjectural; but of the general principle of the desirability of the purchase the Society was fully convinced, and they affirmed it at a Special General Meeting on March 4th, 1889, in the following resolutions, which were confirmed on March 11th.

I.—That the recommendation of the Council to purchase the house, No. 20, Hanover Square, be and is hereby approved by the Fellows of the Royal Medical and Chirurgical Society of London in Special General Meeting assembled; and that steps be immediately taken to carry this recommendation into effect.

II.—That the Council of the Royal Medical and Chirurgical Society of London be and is hereby authorised to do one or all or several of the following acts for and on behalf of the Society, namely:

(1) To acquire the freehold and leasehold property, in the premises at No. 20, Hanover Square, by purchase, for a sum not exceeding £23,000: such property to be vested in the Trustees of the Society.

(2) To lease, sell, or mortgage the leasehold premises now occupied by the Society at No. 53, Berners Street.

(3) To raise such funds as may be required for the acquisition of the premises in Hanover Square; and for such additions and alterations as may be required.

(*a*) By mortgage of the Society's leasehold and of the property to be acquired.

(*b*) Or by the issue of Bonds among the Fellows of the Society.

(*c*) Or by such other means as may seem to the Council most advantageous to the interests of the Society.

(4) To do all such acts and employ such persons as are necessary or advisable for the carrying out of these purposes.

III.—That the Council of the Royal Medical and Chirurgical Society of London be and is hereby authorised to instruct the Trustees of the Society to sell out the securities now vested in their names, and to pay the proceeds of such sale to the Treasurers.

IV.—That the Council of the Royal Medical and Chirurgical Society of London be and is hereby authorised to appoint a Building Committee to complete the purchase of the new premises, and under the direction of the Council to carry out such alterations and repairs as are required therein, and that such Committee consist of the following gentlemen:—Dr. Cheadle, Dr. Gee, Dr. Hare, Dr. Isambard Owen, Mr. Timothy Holmes, Mr. Alfred Willett, Mr. R. W. Parker, and Mr. Warrington Haward; and that the Council have power to add to the number of the Committee if they think it necessary.

The Building Committee so constituted lost no time in commencing operations, and on the very day of its legal constitution issued a circular to the Fellows inviting immediate subscriptions to a proposed Debenture Loan, with a view of paying £10,000 of the purchase-money, in order to secure immediate possession of the premises. Subscriptions were forthcoming immediately, and £7,500 was called up at once, in order (with £3,016 realised by the sale of the Society's investments) to make up the sum required. Immediate possession of the premises was obtained, and the plans for the necessary alterations were at once taken in hand.

The power conferred on the Council by the last of the above resolutions was exercised later on by adding Mr. Clinton Dent to the Committee.

It was soon found that it would be greatly to the advantage of the Society to enlarge the original scheme in three important particulars.

1. The house had a good second floor, but only attics above this, so that the Resident Librarian would have to be lodged on the second floor. By raising the house another storey, and converting the old attic floor into good substantial rooms, an excellent residence has been pro-

vided for the Resident Librarian, and the whole of the second floor has been set free for letting purposes. The cost of this alteration was about £1,400, while the rent of the rooms on the second floor, let to Mr. Belcher and to the Royal Microscopical Society, is £310.

2. It became at once obvious that much more library room must be provided, unless the books were to be kept in the meeting-room, an arrangement which would be very injurious to the pecuniary interests of the Society, as well as very inconvenient, in view of the use expected to be made of the meeting-room for the accommodation of other Societies both during the day and at night. Moreover the use of the meeting-room for library purposes was strongly condemned in a communication from the Council to the Building Committee, April 17th, 1889. Consequently the new library was planned, and thus the greater part of the garden was covered. These various works raised the contract for the new building to £6,840; and even after this contract had been made and the building had been put in hand, the Pathological and Clinical Societies pointed out, with unanswerable force, that their operations could not be carried on without a separate room, in which patients, microscopes, &c., could be exhibited. Hence the north room had to be built, and this again increased the sum payable to the contractor, Mr. Nightingale, which, with all extras and alterations, has amounted to £8,339. The whole of this has been paid with the exception of £39, against which, it is believed, a set-off can be shown.

3. This provision for the library made it necessary to build the meeting-room at the foot of the garden, a small portion of which, and the land behind, occupied by the stables, belongs to the City of London, and was held on a ground-lease, of which thirty-seven and a half years were unexpired, at a rent of £5 10*s*. As it would not have been prudent to erect the meeting-room (which trenches on the leasehold part) on land held for so short a term, the Corporation were approached with the view

of obtaining a longer lease, and, if possible, on more favorable terms than those which they had offered to the Society's predecessors in title, on the ground that the Royal Medical and Chirurgical Society is a scientific body, while they were merely engaged in a commercial venture. The result was that the Corporation most kindly and liberally offered a renewed lease for eighty years at a rent of £30. The formal completion of this lease has been delayed until the new building on the leasehold ground should be finished, but it can be granted almost immediately on the Society authorising its acceptance; and when this is done the Society will have a complete title to the whole property, and the issue of the Debentures for the money borrowed can be proceeded with.

It was soon found that the immediate proximity to the new meeting-room of the old stables (which were in bad repair, and would consequently be a source of expense) would be an inconvenience and annoyance, and the committee consider themselves most fortunate in having been able at once to get rid of a nuisance, and make a most profitable investment for the Society. An agreement has been entered into with Mr. Webb Miles, the owner of adjoining property in Brook Street, by which he undertook to accept a long lease of suitable workshops to be erected for him on the site of the old stables. This improvement has been carried out at a cost not exceeding £1,500, and for the new building Mr. Webb Miles pays us £350 a year, while the old stables only produced a rent of £100. Nor is this the only advantage gained by the change, for it was found possible to reserve a portion of the old stable site, and turn it to most valuable account as an accumulator room.

The enlarged premises which the Society has thus acquired have been found capable of receiving numerous tenants, whose rents will, it is believed, place the Society on a secure financial basis. The following is a list of the present tenants of the Society and their yearly rents:

Messrs. Phipps & Dawson, 53, Berners Street	£450
Mr. Webb Miles	350
Royal Society of Literature	230
Obstetrical Society	200
Mr. J. Belcher, F.R.I.B.A.	180
Royal Microscopical Society	130
Gynæcological Society	126
Mr. Ashdown	120
Clinical Society	105
Pathological Society	105
Royal Historical Society	95
Quekett Club	55
Widows and Orphans Society	50
Library Association	40
British Nurses' Association	25
Lantern Society	25
Royal Agricultural Society	10
Mr. Clinton T. Dent	10
	£2306

The rents received in Berners Street were:

Clinical Society	£75
Pathological Society	75
Widows and Orphans Society	50
Obstetrical Society	42
	£242

The Royal Medical and Chirurgical Society now occupies only the ground floor of the house, the front room on the first floor (which was used at first as the Council room) being let to the Royal Society of Literature, who, however, very kindly allow the Society to use the room for evening Councils and Committees, when necessary. The building of the north room, besides providing a Council room for the Society enables them to receive as tenants the Royal Historical Society, the Royal Society of Literature, the Library Association, the Quekett Club, the

British Nurses' Association, and the Lantern Society, the aggregate rents of which amount to £470.

Another source of extra expenditure is the electric lighting. Before the Society moved it had been quite decided that the electric light should be adopted, but it had not been settled whether the current should be generated by a private installation or obtained from a supply company. A private installation involves a much heavier expense at the outset, but the annual cost is less, while its great superiority consists in the possibility of storage by means of an accumulator, and the consequent freedom from any risk of sudden stoppage or diminution of light. Up to the present time the Committee have every reason to be satisfied with the installation which has been put in by Messrs. Phipps and Dawson, the Society's tenants in Berners Street. The sum of £1,400 has been paid them, leaving a balance still owing of about £150, and a contract has been entered into, by which they undertake for £125 a year to maintain the installation and guarantee an adequate supply of light. To this annual payment, however, something like £45 a year may have to be added hereafter for keeping the accumulator in repair.

It may be mentioned that the cost of the light, if obtained from a supply company would have exceeded £200 a year, exclusive of the cost of maintaining the fittings, lamps, &c.

The question of electric lighting was brought by the Building Committee under the notice of the Council on October 15th, 1889, and the committee were authorised to settle the matter according to their best judgment.

At the same meeting the Council expressed to the Building Committee their opinion that it would be very desirable to add a portico to the house. The committee postponed the question until they had completed the letting of the premises, and a good deal of delay was caused by the London County Council, whose sanction had to be obtained; but the portico is now in course of erection.

The Committee have, in the foregoing statement, endeavoured to give a succinct account of their chief operations down to the present time.

The financial position of the Society is now as follows :— There has been expended, down to the end of the year 1890, £37,008. Of this sum £3,058 was provided by selling the Society's investments, and by interest on deposits at the bank. £30,000 has been received from subscribers to the Debenture Loan at 4 per cent., £1,500 has been temporarily advanced by the President and Treasurers individually at the same interest, and the balance £2,450 is advanced by the Society's bankers on the personal security of Dr. Hare, also at the same rate of interest.

The further liabilities of the Society in respect of the new premises are estimated as follows :

Mr. Nightingale, principal contractor . .	£39
Mr. Robertson (portico, book-cases, extras in Dering Yard, &c.)	406
Messrs. Lake, Beaumont, and Lake, solicitors .	500
Sundry other accounts, not likely to exceed .	800
	£1,745

It will thus be seen that a sum of about £5,700 will have to be raised in addition to the £30,000 covered by the original loan. The Committee therefore propose that a general meeting shall be summoned, and that powers shall be sought to raise an additional £6,000 on Debenture Loan, those powers only to be used to the extent needed to discharge the debts of the Society.

The Society's income and expenditure for 1891 may be thus estimated :

Income.

Subscriptions, fees, &c. (say) .	£1,700[1]
Rents	2,306
	£4,006

[1] The receipts of the year 1889 were £1,481 ; those of the year 1890 were £1,642.

There will also be an additional income from occasional lettings of the two meeting-rooms.

Ordinary Expenditure.

Rent of leaseholds (Hanover Square and Berners Street)	£44	
Rates and taxes	188	
Electric lighting	170	
Water	50	
Insurance	30	
Coals and gas	70	
Furniture and repairs	40	
Meeting expenses	40	
Transactions and *Proceedings*	350	
Printing and stationery	80	
Salaries and wages	670	
Library	300	
Miscellaneous incidental expenses	100	2,132

Extraordinary Expenditure.

Annual sum to be devoted to payment of Interest and Extinction of Debt (calculated by the Society's Accountants to pay off the whole of the Debentures in forty-eight years)	[1] 1,700	
Balance available for contingencies	174	
		£4,006

A probable balance of £174 is thus left over at the end of the year 1891, but it is necessary to bear in mind that the annual expenditure is likely to increase in future years because of a larger outlay for repairs. The Council, however, is of opinion that the receipts of the Society will probably increase at an equal or an even larger rate

[1] At end of first year the interest will be £1,440, and £260 will be paid to the Trustees of Debenture-holders—in succeeding years the amount of interest will fall, as that of the repayment rises.

by increase in the number of Fellows, and the more frequent letting of the meeting rooms.

It must not be forgotten, however, that the Society will lose £435 a year on the termination (fourteen years hence) of the Berners Street lease ; so that it would be right to apply during the interval as much as possible of the profit rental obtained from the Berners Street premises to leave the Society in as good a position on the termination of the Berners Street lease in 1904 as it is at present.

The capital expenditure having been thus increased beyond what was originally contemplated, there remains the question how to provide the funds.

There are only two courses open : (1) to raise the sum on Debentures, or (2) to treat it as a floating debt, and discharge it gradually out of the Society's surplus income. The second course involves the payment of a larger interest, for bankers are not likely to lend at so low a rate as 4 per cent. for an unlimited time. This would diminish for a considerable period the amount annually applicable to the extinction of the debt. In all respects, therefore, it seems better to authorise an addition, within the limit of £6,000, to the amount of the original Debenture Loan, nor does there seem any difficulty in the way. The present limit of £30,000 was fixed by the committee of the subscribers to the loan on April 16th, 1889, at a time when much less expensive building operations were contemplated,[1] and when the security for the loan was therefore much less than at present, but the committee under the authority of the Council felt justified in incurring a larger expenditure on the grounds previously stated, and it has therefore been decided under the advice of the Society's solicitors, to ask the Council to summon a General Meeting of the Society : (1) To approve and accept the lease from the Corporation of the City of

[1] A resolution stands on the minutes of the Building Committee, April 5th, 1889, limiting the amount to be spent on building to £4,500, and the total expenses to £6,000.

London, and (2) to sanction the proposed extension of the loan ; to be followed (after passing the second of these resolutions) by a meeting of subscribers to the loan for the same purpose.

All the members of the committee appointed at the meetiug of subscribers to the Debenture Loan held on 16th April, 1889 (for the purpose of settling and approving on behalf of the intended Debenture holders the form of Debenture and Trust Deed) have, at a meeting between themselves and the President and Treasurers of the Society held on 11th April, 1890, expressed their individual assent to an extension of the total amount of the Debenture Loan to £36,000, although (having regard to what has been said in previous circulars and at previous meetings of subscribers to that Loan) they prefer, notwithstanding the powers conferred on them at the meeting of 16th April, 1889, to have the assent of a further meeting of their constituent body to the proposed extension.

The Resident Librarian has already received applications for additional Debentures in excess of the sum required.

As soon as the limit of the loan has thus been definitely fixed, and the new lease from the City granted, the Deed of Security for the benefit of the Debenture-holders can be completed and the Debentures issued. The forms of both of these have been long ready.

When the few remaining additions to the building are completed, and the accounts are settled, the functions of the Building Committee will have ceased. Their labours have not been light, for down to the end of the year 1890 seventy-seven meetings have been held, but they have been most interesting, and, the Committee hope, have resulted in lasting advantage to the Society.

The complicated transactions confided to the committee could not have been successfully conducted without efficient professional assistance ; and it would be an unpardonable negligence not to record the deep debt of gratitude which

the Society owes to Mr. William Flockhart, the architect, under whose advice the buildings have been completed, and to Mr. Thomas Beaumont, the solicitor, by whom the numerous, and in some cases difficult, legal questions involved have been settled to the best advantage of the Society. The Council is already aware that in every detail of the Committee's work, from the least to the greatest, the zeal, energy, and ability of Mr. MacAlister, the Resident Librarian, have rendered services which are simply invaluable.

Signed for the Committee,

TIMOTHY HOLMES,
Chairman.

SPECIAL GENERAL MEETING,

Wednesday, February 18th, 1891.

At 5 o'clock p.m.

Timothy Holmes, F.R.C.S, President, in the Chair.

Frederick Taylor, M.D., }
Warrington Haward, F.R.C.S., } Hon. Secs.

Present—29 Fellows.

The President called upon Mr. Haward (Honorary Secretary) to read the circular convening the meeting.

The Report[1] of the Building Committee as adopted by the Council being taken as read, the President moved, and Mr. Bostock seconded :

(1) That the Report of the Building Committee to the Council, on January 21st, 1891, adopted by the Council on January 24th, 1891, and the action of the Committee and Council respectively with refer-

[1] See p. lxxxvii.

ence to the various matters stated in that Report, be hereby confirmed and adopted by the Society; and, in particular, that the sanction of this meeting be and is hereby given to the granting of the various Leases mentioned or referred to in the said Report, and to the acceptance of the new lease from the City of London therein referred to, and to the previous surrender to the City of the existing Lease; and that the Council of the Society be and is hereby authorised to execute all such leases, deeds, and other documents as may be necessary or expedient to carry the various arrangements mentioned in the said Report with reference to the Society's property into effect.

Mr. Cripps criticised the Report, and stated that as a member of Council he did not agree with it, and Mr. Bruce Clarke moved the following amendment:—"That the Council be requested further to consider the Report of the Building Committee (and the other matters contained in the Council resolutions) more especially with regard to the question of further increasing the Society's income which is not at the present time adequate to the claims that are likely to be made upon it." This was seconded by Mr. Butlin.

Questions were asked by various Fellows and Mr. Beaumont, the Society's Solicitor, was asked by the President to answer such as came within his department.

The President then answered *seriatim* the various points raised, and the amendment being put to the meeting was negatived.

The original resolution was then put and carried *nem. con.*

The President then moved, and Dr. Hare seconded:

(2) That for the purpose of carrying out such arrangements, and for the other purposes of the Society, the Council of the Society be and is hereby authorised to do all or any one or more of the following acts or things for and on behalf of the Society, viz.:

(*a*) To raise any sum of money not exceeding in the whole (including the sum of £30,000 already received from Subscribers to the intended Debenture Loan) the sum of £36,000, by accepting such offers of Fellows of the Society willing to contribute as the Council may think proper:

(*b*) To secure the principal sum for the time being raised, with interest at the rate of 4 per cent. per annum payable half yearly, by the issue of Debentures to the Fellows of the Society contributing to the Loan; and, further, to secure the payment of the principal and interest for the time being payable on such Debentures or on any added or substituted Debentures, by a Trust Deed to be made between the Society of the one part, and Dr. Thomas Barlow, Dr. Samuel Jones Gee, Dr. Charles Theodore Williams, and Mr. John Warrington Haward (nominees of the intended Debenture-holders), as Trustees for them, of the other part, in such form as shall be approved by the Committee appointed for that purpose by the subscribers to the proposed Debenture Loan at a meeting of such subscribers held on 16th April, 1889;

(*c*) To distribute the Debentures among the contributing Fellows in such manner as the Council may deem just; and

(*d*) To do whatsoever else the Council may think fit or be advised for effecting any purpose hereby authorised.

This was carried *nem. con.*

Sir Edward H. Sieveking moved, and Dr. Blandford seconded:—"That a very hearty vote of thanks be offered to the President and to the other members of the Building Committee for their valuable services to the Society." This was put to the meeting by Sir Edward Sieveking, and carried by acclamation.

ANNUAL GENERAL MEETING.

Monday, March 2nd, 1891.

At 5 p.m.

Timothy Holmes, F.R.C.S., President, in the Chair.

Frederick Taylor, M.D.,
Warrington Haward, F.R.C.S., } Hon. Secs.

The Minutes of the Special General Meeting of February 18th were read and confirmed.

The President nominated Dr. Mapother and Mr. R. W. Parker to act as Scrutineers of the Ballot for the election of officers and council for the ensuing year.

The President then called upon Mr. Haward (Honorary Secretary) to read the Report of Council, which was as follows :—

"The Report presented to the Society at the last Annual Meeting gave an account of the considerations which led to the determination of the Society to move from the

premises which it had occupied at 53, Berners Street since the year 1834, to the present house.

"It also described the steps that were taken, in accordance with a series of resolutions passed at a Special General Meeting of the Society on March 4th, 1889, to acquire the premises at 20, Hanover Square, to make such alterations and additions as were required, and to raise the necessary funds. At the same General Meeting a "Building Committee" was appointed to carry out the business which these measures involved. The work of the Building Committee, so far as it had then proceeded, was described in the same Annual Report (see *Transactions*, vol. lxxiii), and the one presented by the Building Committee to the Council on January 24th, 1891, brings its record up to the present time.

"It was thought desirable by the Council to bring this Report under the consideration of a Special General Meeting of the Society, which was held on February 18th, at which the following resolutions were passed :

"(1) That the Report of the Building Committee to the Council, dated January 21st, 1891, and adopted by the Council on January 24th, 1891, and the action of the Committee and Council respectively with reference to the various matters stated in that Report, be hereby confirmed and adopted by the Society; and, in particular, that the sanction of this meeting be and is hereby given to the granting of the various Leases mentioned or referred to in the said Report, and to the acceptance of the new lease from the City of London therein referred to, and to the previous surrender to the City of the existing Lease; and that the Council of the Society be and is hereby authorised to execute all such leases, deeds, and other documents as may be necessary or expedient to carry the various arrangements mentioned in the said Report with reference to the Society's property into effect.

"(2) That for the purpose of carrying out such arrange-

ments, and for the other purposes of the Society, the Council of the Society be and is hereby authorised to do all or any one or more of the following acts or things for and on behalf of the Society, viz.:

(*a*) To raise any sum of money not exceeding in the whole (including the sum of £30,000 already received from Subscribers to the intended Debenture Loan) the sum of £36,000, by accepting such offers of Fellows of the Society willing to contribute as the Council may think proper:

(*b*) To secure the principal sum for the time being raised, with interest at the rate of 4 per cent. per annum payable half yearly, by the issue of Debentures to the Fellows of the Society contributing to the Loan; and, further, to secure the payment of the principal and interest for the time being payable on such Debentures or on any added or substituted Debentures, by a Trust Deed to be made between the Society of the one part, and Dr. Thomas Barlow, Dr. Samuel Jones Gee, Dr. Charles Theodore Williams, and Mr. John Warrington Haward (nominees of the intended Debenture-holders), as Trustees for them, of the other part, in such form as shall be approved by the Committee appointed for that purpose by the subscribers to the proposed Debenture Loan at a meeting of such subscribers held on 16th April, 1889;

(*c*) To distribute the Debentures among the contributing Fellows in such manner as the Council may deem just; and

(*d*) To do whatsoever else the Council may think fit or be advised for effecting any purpose hereby authorised.

"It will therefore be seen that the Building Committee have almost completed the work for which they were appointed, with results which the Council believe will be

considered by the Fellows as eminently satisfactory, and that the Committee will soon be dissolved.

"The Council have the pleasure of acknowledging the very generous gift by the President, Mr. Timothy Holmes (who has also throughout been Chairman of the Building Committee) of the handsome and convenient presidential table, now in the Society's meeting-room.

"The Council are glad to announce also the gift of £100 from Dr. R. Douglas Powell to the Permanent Endowment Fund, being the amount of his subscription to the Debenture Loan. The amount of the Fund is now £374.

"Particulars of the income and expenditure of the Society during the year 1890 are set forth in the Treasurers' statement (page 88).

"Thirty-seven new Fellows have been elected during the twelve months from March 1st, 1890, to February 28th, 1891, of whom thirty-two are resident and five non-resident. One Fellow has resigned, and nineteen have died during the same period. The total number of Fellows on the List is now 778.

"In view of the somewhat altered circumstances of the Society, the Council have thought it desirable to subject the Bye-laws to revision; and a Committee was recently appointed to consider and report upon this matter. In accordance with the recommendations of that Committee, the Council propose to the Society the following alterations in the Bye-Laws:

In Chapter II, Section I, lines 16 and 17, the word "Library" shall be substituted for the words "Common Meeting Room."

In Chapter IV, Section II, line 10, the word "Library" shall be substituted for the word "Room."

In Chapter V, the whole of Section II shall be expunged, and the following Section shall be substituted therefor, viz.:

"II. All sums of money paid to the Society as Composition fees in lieu of Annual contributions shall be applied in reducing the Principal money owing upon the

Debenture loan of the Society, and shall be paid by the Treasurers to the Trustees for the Debenture holders as and when the Council shall from time to time direct."

In Chapter V, Section IV, line 3, the words "Society's Library" shall be substituted for the words "Public Meeting Room."

In Chapter V, Section V, line 7, the word "full" shall be inserted between the words "the" and "privileges;" and in line 9, the words "any non-resident Fellow wishing to borrow books from the Library shall upon paying the Annual Contribution of One Guinea be allowed to have one volume at a time," shall be inserted after the word "guineas."

In Chapter IX, Section V, the words commencing with the word "Committee," in line 2, shall be expunged, and the words "Chartered Accountant to be appointed by the Council" shall be substituted therefor.

Section VI shall be expunged, and the following Section shall be substituted therefor, viz.:

"VI. The Accounts so audited shall be laid before the Council at their meeting in February, and shall be presented to the Society at the Annual General Meeting."

In Chapter X, Section II, line 1, the words "shall be *ex-officio* members of all Committees and" shall be inserted between the words "Secretaries" and "shall."

In Chapter XIII, Section II, line 2, the words "except the months of July, August, and September, and the Tuesday in Easter week," shall be inserted between the words "month" and "or."

In Chapter XIV, Section II, line 2, the word "the" shall be substituted for the word "an," and the words "of Three Guineas" shall be inserted between the words "contribution" and "whose," and in line 6 the words "or in case of Non-resident Fellows his commencing to pay the annual contribution of Three Guineas" shall be inserted between the words "Fellow" and "and."

In Chapter XV, Section VI, line 2, the word "Library" shall be substituted for the word "rooms."

In Chapter XVI, Section II, line 1, the words "of Three

Guineas" shall be inserted between the words "contribution" and "or"; and in line 5 the words "Any non-resident Fellow paying the annual contribution of One Guinea shall be allowed to borrow books from the Library, but shall not have more than one volume at a time" shall be inserted between the words "time" and "pamphlets."

In Chapter XVII, Section I, line 8, the following words shall be inserted after the word "meet," viz. "nor on the usual Tuesday if it shall fall in Easter week, in which case there shall be a meeting on the fourth Tuesday in June."

In Chapter XVIII, Section III, line 2, the word "Library" shall be substituted for the word "room."

In Chapter XIX, the following shall be substituted for Section I:

"I. The whole of the Society's property and effects, of what kind soever, shall be under the direction, management, and control of the Council; but the Council shall not sell or mortgage any of the Society's lands, tenements, or hereditaments without the sanction of a Special General Meeting of the Society or of the Annual General Meeting, due notice having been given of the business to be then taken into consideration."

"The Honorary Librarians report as follows:

"We are pleased to report that the year's experience of the new Library rooms amply justifies the hope we expressed last year that the acquisition of the new premises would be an especial benefit to the Library. It was impossible that such a change could have been effected, involving as it did the redistribution of nearly 40,000 volumes, in rooms differing in every way from those at Berners Street, without for some time interfering considerably with the work of the Library and the convenience of Fellows, and we thankfully acknowledge the kindly consideration and forbearance displayed by the users of the Library during a

period which to them must have been extremely uncomfortable. Our small staff had so many other duties thrust upon them in connection with the removal, that for some time it was impossible for them to give the same attention as formerly to the Library, and this also made a considerable demand upon the forbearance of readers. Under the circumstances, however, we think the Society may fairly congratulate itself that the discomfort and inconvenience have not been greater, and we believe that the new rooms and the arrangements generally give entire satisfaction to the Fellows who use them.

"The heavy strain upon the funds of the Society has compelled us to spend a smaller amount than usual upon the purchase of books, but all periodicals, series, academies, and works in continuation have been kept up to date; and we have endeavoured as far as possible to procure for the Library every book which has been specially recommended to us by the Fellows, as well as many others of importance which the Library Committee have ordered from time to time, and we are happy to say that the binding has not been allowed to fall into arrear.

"The Resident Librarian had, before leaving Berners Street, begun the preparation of a new Supplementary Catalogue, and it had already reached the proof stage when the warehousing of the books put a stop to the correction of the proofs. This work can now be pushed on, and we trust that the Catalogue will be in the hands of the Fellows very shortly.

"The dwarf bookcases which have been lately placed in the corridor will for a time be partly used for the libraries of some of our tenant Societies, but will ultimately be occupied by our own books, and this latest addition to the shelving accommodation,

together with other shelving at present unoccupied, will provide for the needs of our Library for many years to come."

(Signed) SAMUEL JONES GEE, J. W. HULKE. } *Hon. Librarians.*

"The Committee appointed to investigate the climatology and balneology of Great Britian and Ireland, have been making satisfactory progress with the work before them; but, owing to the very large amount of information to be collected and dealt with, it is not as yet possible for anything of the nature of a preliminary report to be issued.

"The climatological portion of the work is being carried out by means of a series of inquiries addressed to medical men practising in various parts of the kingdom, by the study of published meteorological records, and where possible, by personal visits of members of the Committee to the various health resorts. The balneological inquiry is being conducted upon somewhat similar lines. Requests for information upon certain points were addressed to practitioners at the important bath places, all of which will be visited, and some of which have been already visited and reported upon by members of the Committee. The Committee will gladly welcome any information which may be offered to it bearing upon the subjects under inquiry. Such information should be addressed to the Honorary Secretary.

"Another Committee have recently been appointed to consider the treatment of suspended animation in the drowned.

"In conclusion the Council wish to acknowledge the great assistance they have received in every department of the Society's work from the Resident Librarian, Mr. J. Y. W. MacAlister.

(Signed) T. HOLMES, *President.*"

Dr. Hare (Treasurer) then read the Annual Statement of Accounts as audited (see pp. cxiv, cxv).

Sir Edwin Saunders said that as a comparative stranger, though once much more at home in the meetings of the Society, he could not forbear to congratulate the Society upon their excellent premises, which were admirably adapted to their requirements. He thought that the action of the Building Committee was entitled to the greatest praise. They all knew how architects and builders were apt to go wrong unless looked after. He had been struck by the elevation and the lighting of the rooms; and he must add a word of praise for the handsome desk, the present of their President. He had great pleasure in moving: "That the Report of the President and Council together with Treasurers' audited Statement of Accounts be adopted, and they are hereby ordered to be printed in the next volume of *Transactions*." This was seconded by Mr. George Pollock and carried *nem. con.*

Dr. Hare then moved: "That the very hearty thanks of the Society be given to Sir Richard Quain, Mr. Hussey, Dr. Douglas Powell, and Mr. E. U. Berry for their generous donations to the Permanent Endowment Fund." He thought it was a great thing for the Society to have established such a fund, which would ultimately give great steadiness and support to its finances. Although it had only been established a little over twelve months, they had had contributors from two branches of the profession. Sir Richard Quain had given £100, Mr. Hussey £50, Dr. Douglas Powell £100, and Mr. Berry had made an arrangement, which it was unnecessary to describe, but which had resulted in a net gift of £124 2*s.* to the Society. These various sums were invested in New South Wales Stock, and brought in an annual interest of £13. The Treasurers entertained strong hopes that the contagion might spread, and that gentlemen who were tired of receiving trifling dividends of £4 or less would divest themselves of the responsibility by handing over their debentures to the fund. A good deed would be done to

the Society, and he hoped the time was not very far distant when the income arising from this endowment fund, which could not under any circumstances be alienated, would form an important source of revenue, which would prove a lasting benefit to the Society.

Mr. Bostock endorsed the remarks of his senior colleague and seconded the motion, which was agreed to *nem. con.*

The President then moved: "That the alterations of the Society's Bye-Laws, as set forth in the Council's Report, which, in pursuance of the provisions of the Charter, have been made by the Council, be and are hereby confirmed." He explained that it had become necessary to adjust the laws to the altered circumstances in which the Society now found itself. Many of the changes were purely technical; and he suggested that it would be an economy of time to ballot for them *en bloc*, unless any Fellow wished any one of them to be voted on apart. He pointed out that a majority of not less than two thirds of those present was necessary.

Mr. Hulke seconded the motion, adding that the proposed alterations had received the most careful consideration at the hands of a committee specially appointed for the purpose.

The various alterations were then balloted for *en bloc*, and were declared by the President to have been duly carried.

The President then delivered his Annual Address.

Sir Edward Sieveking said they had at first to complain of a disagreeable echo in their new meeting room, which was now happily banished; but he hoped he would not be complained of if he played the part of an echo to the President's most excellent Address. It was a distinct improvement on the past practice to shorten the obituary notices for the meeting, but they would be grateful if he would allow the fuller notices of the distinguished men who had passed away during the last year to appear in the *Transactions*. The trouble and pains which the President took in writing his address justified its being

published, in order that what had been done during the various years might be placed on record. The address was so full of suggestion that it would take up too much time to discuss the points in detail, but they ought to be considered *seriatim* by the Council and brought before the Society later on. He confessed he had never listened to an address so full of matter and so likely to be of use in promoting the welfare of their Society. He, therefore, wished to move "that the best thanks of the Society be given to the President for his address, and that he be requested to allow it to be printed."

Mr. Thomas Smith said as that, as a Fellow of the Society and as an old friend of their President, he had great pleasure in seconding the motion. The President had alluded to many interesting points, which it was not opportune just then to discuss; but there was one point to which he felt he must allude, that was the possible development of the Society in a social direction. While the time might not yet have arrived for an amalgamation of all the learned societies of the metropolis (personally he did not think it would ever come), it would be an enormous advantage to this Society if it could be developed on the lines suggested by the President.

The motion was carried by acclamation.

The President thanked the Fellows for their kind remarks, and promised to lay before the Council the obituary notices, which he had prepared for publication, and he hoped that personal friends of the deceased gentlemen would take advantage of the opportunity to fill up any *lacunæ*.

Mr. Bostock called attention to the paragraph in the report, informing them that the magnificient table which they must all have admired had been presented to them by their President. He moved: "That this meeting desires to offer its cordial thanks and to record the Society's indebtedness to the President, Mr. Timothy Holmes, for the handsome and valuable gift of the presidential table which had been placed in the meeting-room."

Mr. Alfred Willett seconded the motion, observing that the design of the desk was artistic, convenient and dignified. He asked permission to add to the motion a proviso that some memorial of the fact of the donation should be placed on a suitable part of the desk.

Agreed to *nem. con.*

On the motion of Dr. George Harley, seconded by Mr. J. H. Morgan, it was resolved: "That the best thanks of the Society be given to the retiring Vice-Presidents, Dr. Robert Barnes and Mr. Croft, for their services to the Society during their term of office."

On the motion of Sir Richard Quain, seconded by Mr. Howard Marsh, it was resolved: "That the best thanks of the Society be given to the retiring Members of Council, Dr. Ord, Dr. Gilbart Smith, Mr. Clinton Dent, and Mr. Howse, for their services to the Society during their term of office."

The Scrutineers then presented their Report, and the President announced that the following gentlemen had been duly elected as Officers and Members of Council for the ensuing year:—

President.—Timothy Holmes.

Vice-Presidents.—J. Langdon Down, M.D.; George Harley, M.D., F.R.S.; F. Howard Marsh; Alfred Willett.

Treasurers.—Charles John Hare, M.D.; John Ashton Bostock, C.B.

Honorary Secretaries.—Frederick Taylor, M.D.; J. Warrington Haward.

Honorary Librarians.—Samuel Jones Gee, M.D.; John Whitaker Hulke, F.R.S.

Members of Council.—Walter Butler Cheadle, M.D.; W. R. Gowers, M.D., F.R.S.; Norman Moore, M.D.; John Williams, M.D.; George Vivian Poore, M.D.; William Harrison Cripps; Charles N. Macnamara; Henry Walter Kiallmark; Herbert William Page, M.C.; Knowsley Thornton, M.B., C.M.

		£	s.	d.	£	s.	d.
	DR.						
To Balance in hand on January 1st, 1890 :							
Cash in hand		33	3	8			
„ at Bankers		971	19	6			
„ on Deposit		300	0	0			
					1305	3	2
368 Annual Subscriptions at £3 3*s*.		1159	4	0			
45 Entrance Fees at £6 6*s*.		283	10	0			
3 Composition Fees for *Transactions* at £8 8*s*.		25	4	0			
7 „ (Life)		124	19	0			
Fines		0	4	6			
					1593	1	6
„ *Sales :*							
By Messrs. Longmans (*Transactions*)		45	13	1			
„ Librarian (*Proceedings*)		0	10	4			
„ „ (Catalogue)		1	10	0			
					47	13	5
„ Rents and Contributions from Societies, &c.					1157	13	1
„ *Interest :*							
On Deposit Account		3	15	10			
„ Permanent Endowment Fund		2	11	0			
From Prudential Insurance Co.		8	9	8			
					14	16	6
					£4118	7	8

CHARLES J. HARE,
J. A. BOSTOCK, } *Treasurers.*

PERMANENT

	£	s.	d.
DR.			
To Sir Richard Quain's Donation	100	0	0
„ Mr. Hussey's Donation	50	0	0
„ Dr. Douglas Powell's Donation..	100	0	0
„ Mr. Berry's Donation	124	2	0
	£374	2	0

MARSHALL

	£	s.	d.
DR.			
By Dividends for 1890	21	18	8

[Amount of "Consols" on December 31st, 1889....£616 4*s*. 2*d*.]

		£ s. d.	£ s. d.
Cr.			
By Rent, Rates, Taxes, &c.		..	115 7 1
„ Lighting, Cleaning, and Heating		..	73 9 4
„ Repairs, Furniture, &c.		..	160 11 7
„ Meeting Expenses		..	49 2 10
„ *Transactions* and *Proceedings*		..	398 12 6
„ Stationery and Sundry Printing, Stamps, &c.		..	112 14 7
„ Salaries and Wages		..	704 6 0
„ Library, Books, and Binding		..	139 11 2
„ *Extraordinary Charges:*			
"Spas" Committee		16 18 2	
Messrs. Cooper Bros. } Accountants		10 10 0	
Messrs. H. W. Kirby & Co. }		2 2 0	
„ Honorarium to Resident Librarian		105 0 0	
„ Loan to Building Account (Balance)		477 3 1	
			611 13 3
„ Repayment of Amount due on account of Petty Cash ..		..	31 10 11
„ Interest on Debentures:			
For broken period ending January 1st, 1890		668 17 4	
For six months ending July 1st, 1890		544 12 1	
			1213 9 5
„ Annuity		..	34 16 0
„ Cheques, &c.		..	0 8 5
„ Sundry Payments (Petty Cash)		..	20 0 6
„ *Balance:*			
In hand £27 15*s.* 9*d.* At Bank £424 18*s.* 4*d.*		..	452 14 1
			£4118 7 8

Audited and found correct, 16th February, 1891.

HERBERT W. PAGE. | C. B. LOCKWOOD.
DAVID W. FINLAY. | ALFRED WILLETT.
A. PEARCE GOULD. |

FREDERICK TAYLOR, *Hon. Sec.*

ENDOWMENT FUND.

	£ s. d.
Cr.	
By Purchase of £326 7*s.* 3*d.* New South Wales 4 per Cent. Stock	373 17 0
„ Power of Attorney for Bankers	0 5 0
	£374 2 0

CHARLES J. HARE, } *Treasurers.*
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HALL FUND.

	£ s. d.
Cr.	
By Purchase of £21 3*s.* 10*d.* "Consols"	21 18 8

[Amount of "Consols" on December 31st, 1890....£637 8*s.*]

CHARLES J. HARE, } *Treasurers.*
J. A. BOSTOCK, }

ADDRESS

OF

TIMOTHY HOLMES, F.R.C.S.,

PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 2ND, 1891.

GENTLEMEN,—It has always been customary at our Annual Meeting, so long as I have known the Society, for the President to make allusion to the Fellows who have died during his year of office; and this has now grown into so much detail that the Annual Address has become little more than a necrology, the general affairs of the Society occupying a very subordinate place in it. I trust I shall not be suspected of irreverence or ingratitude to the dead, if I reverse this proportion of space—at least in my spoken address, and limit myself here to a more cursory mention of our deceased colleagues. I have prepared a longer obituary notice of each for insertion in the Transactions, if the Council think it worthy of a place there, and it is in our Transactions that these obituary notices have a real and permanent value. Our series, of now over seventy volumes, would have been even more valuable and interesting than it is, if it had contained a short and reliable contemporary notice of all the men who died while Fellows of this Society since the year 1806;

and I much regret that the notices compiled by former Presidents are no longer accessible.

The President's Address was first inserted in our Transactions in vol. lxvi, which commences with the address of the late President who has just passed away—Mr. Marshall. Previous to this very beneficial change the President's Address was published in the Proceedings, but the earlier addresses before the commencement of the Proceedings could now only be discovered by a laborious search through old medical newspapers, where they are probably reported only in abstract.

The number of Fellows who have died since the last Annual Meeting is nineteen. Three of these were Honorary Fellows, one of them Foreign, and two English; of the other sixteen, five were non-resident, and eleven resident Fellows. In the spoken address, I enumerated these in the order of their seniority and of their services to our Society; but in this place it will, I think, be better to commemorate them in the order of their deaths.

Dr. Ainsworth, of Manchester, a cousin of W. Harrison Ainsworth, died on March 6th, 1890, at the age of eighty-seven. He was one of our oldest Fellows, and had earned the respect of his fellow-citizens by a long course of public services. His medical education had been liberal and extensive, as he took the degree of M.D. at Berlin and travelled over most of the countries of Europe. His father was a member of the medical profession, and especially known for his love of botany and natural history, which Dr. Ainsworth also shared. Dr. Ainsworth was never married, and I am not aware of any contribution of his to medical literature.

Dr. Humby was a member of the medical school of St. George's Hospital, and was a well-known and active Fellow of our Society. He served on the Council during the years 1866–7, and few of our Fellows were more keenly interested in the building scheme and the debenture loan, to which he was a large subscriber. He practised for more than half a century in the neighbourhood

of St. John's Wood, and was therefore well known to most of us, and he was a man who gained the esteem of all who knew him. He had only recently retired from practice when his health failed suddenly, and his end was hastened by an attack of influenza complicated with bronchitis. He died on March 27th.

Mr. Gregory Forbes was a respected and eminent general practitioner for many years in the West of London. He was named after the celebrated Dr. Gregory, his great-uncle. He had retired from practice for some time. His health seems to have been shattered by the loss of his wife a few months before his own death, and he was seized with apoplexy, which proved speedily fatal, while on a journey to visit her grave. A large circle of friends and patients can witness to the respect with which he was regarded.

Mr. Gregory Forbes had been a Fellow for more than forty years, had served on the Council and on the Library Committee, and contributed two papers to the Transactions, one in 1849 on a case of impaction of a piece of bone in one of the chief branches of the right bronchus, the other in 1850 on a case of inversion of the uterus. Both are valuable practical papers, the symptoms being well and clearly described, and the treatment intelligently discussed.

A perusal of these early papers causes great regret that the discussions which followed on them are not preserved. We can hardly exaggerate the interest with which we should have read the opinions of the surgeons of that day on the feasibility of removing a foreign body from the pulmonary part of the bronchus itself, an operation which has lately been so successful in the skilful hands of Mr. T. Smith and Mr. Bryant ('Trans.,' vols. lxxi and lxxii). So, again, the discussion which must have followed on the propriety of removing the inverted uterus would have been most interesting and instructive.

Dr. W. H. Holman, of South Hampstead, had long been a Fellow of our Society, but his absorption in the cares and labours of a large general practice seems to have prevented him from taking any share in the Society's

proceedings. His father was a highly respected practitioner in the East of London, whose uncle, Lieutenant Holman, is celebrated as "The blind traveller." Dr. W. H. Holman, after filling the office of house-surgeon at the London Hospital and obtaining the double qualification, served as house-surgeon to a country hospital (Aylesbury) for a year, and then passed the examination at the University of London and took the degree of M.B. in 1847. About this time he settled in Adelaide Road, South Hampstead, where he carried on a large and successful practice to within a short period of his death, which occurred in April, 1890, in his sixty-sixth year. He was beloved and lamented by a large circle of friends and patients.

Dr. A. Julius Pollock died on May 11th, 1890, after a very few days of serious illness, at the comparatively early age of fifty-five. He was at the time of his death serving for the second year on our Council, and although he had not contributed any paper to our Transactions, he showed his interest in our Society, and rendered us most valuable service by his attention to the duties of the Council, doubly important at this period of the history of the Society, and by taking part in various of its committees. Dr. Pollock was regarded as one of the chief supports of the school of Charing Cross Hospital, over which he had presided for five years as Dean, and where he held the office of Lecturer on Medicine up to the time of his lamented death. He had the reputation of a sound and judicious physician, and his work on rheumatism testifies to these qualities. He was a son of the late Lord Chief Baron of the Exchequer, and a member of a family which has many connections with our profession and with the Royal Medical and Chirurgical Society. Though his death was at last almost sudden (for he had been at work five days before), yet to his friends it was not wholly unexpected, as he was known to be suffering from grave renal disease. An accidental attack of influenza still further depressed him, and this was followed

after a considerable interval by the acute pleuro-pneumonia which proved fatal.

Dr. Walter Pearce had only very recently joined our Society, and was just entering on a career of professional distinction, when a terrible calamity, the result of acute brain disease, deprived the profession of one who would have been one of its chief ornaments, and a large circle of friends of one who was esteemed and loved wherever he was known. He died at the early age of thirty-six. Dr. Pearce had enjoyed a liberal education in many branches of knowledge, having studied in the Royal School of Mines, and taken the degree of Bachelor of Science at the London University before entering on the study of medicine. He had also travelled much and seen life under various aspects. He was connected as Assistant Physician with St. Mary's Hospital, had filled the office of Medical Superintendent there, and was acting as Medical Tutor at the time of his death. He was eager in the cause of temperance, was ardently interested in the volunteer movement, and was indefatigable in the pursuit of clinical medicine, having published many tracts and observations of great practical value, some only a short time before his death. He had only been married a few months, when the awful catastrophe occurred which closed a prospect of so much happiness and usefulness.

Dr. Fincham was an old member of the Society, and had served on the Council. He was long connected with the Westminster Hospital, where he retired from active service in the year 1883. He was a member of the University of Oxford, and received his medical education in England at St. George's Hospital, though he also studied in France and in Ireland. He was a man of retiring habits, but of agreeable and friendly manners; well read in general as well as medical literature, a sound and good physician, and an honorable and respected gentleman, one who worthily maintained the high character of the physician's calling, both in professional and in general circles. He died at the age of seventy-two.

There were few more interesting or more lovable persons in our profession than *Professor William Kitchen Parker*, who was one of our Honorary Fellows, and who died on July 3rd, 1890. Professor Parker was of humble origin, the son, I believe, of a small farmer. This was a fact of which he was so far from being ashamed, that I have heard him refer to it in one of his lectures at the College of Surgeons, and say that he was probably the only member of the College who had himself held the plough; but his want of early literary training was a certain disadvantage to him as a teacher in after life, as it deprived his lectures of that condensation and lucidity which are the best fruits of scholarship. But if Mr. Parker was not "a scholar" in the narrow sense, no man had wider scholarship or more intimate knowledge of the subject to which he had devoted his life—viz. zoology—and in which his name and works have become the common heritage of all nations and of all time. For Professor Parker, though he was fertile and ardent in constructing and following out theories, found his chief pleasure and obtained most of his fame in the laborious demonstration of facts, and these he established on so solid a basis of proof, that no subsequent discoveries or changes of view can interfere with them. He commenced life in London as a Licentiate of the Society of Apothecaries in the year 1849, and for a long series of years he toiled on to support himself and his family by the drudgery of an obscure practice, while giving up every hour of the early daylight or of the intervals which he could snatch from business to the labour of the scalpel and the microscope, by which he followed out the structure and the development of so many classes of animals. At length the Council of the College of Surgeons wished to secure his services as a Lecturer on Comparative Anatomy, and Mr. Parker submitted to the examination and obtained the Diploma of the College in the year 1873. He lectured as Hunterian Professor in the following year, alone during the absence from England of Professor Flower, and after-

wards in conjunction with him for many years. His lectures were an astonishing display of learning gathered through long days of patient labour, and poured out from a mind so full of its subject that one detail suggested another in a way which might well bewilder even a moderately well-informed listener. But if his lectures were sometimes too deep or too full of detail for his audience, the works founded on these lectures, as well as the earlier labours which first introduced him to fame as a zoologist, will long remain as the greatest contribution of our day and country to the science to which he devoted himself.

In the earlier years of his life Mr. Parker struggled on as best he could (and only a man of his vigour and enthusiasm could have maintained the struggle at all) to prosecute his favourite studies, while earning enough to keep his household together. When the value of his work became known he received assistance from the Royal Society, as administrators of the public grant for advancing research, and latterly a small pension on the Civil List enabled him to retire from practice, and in the solitude of the country which he loved, follow the studies in which he found his chief happiness. Unfortunately, his health was not very robust, and he did not live long to enjoy his retirement. He transmitted his tastes to two of his sons, who hold professorships in zoology at Otago in New Zealand, and in the University College of North Wales.

Professor Parker's career has been used as an illustration of the neglect of science by our Government, and as "a serious blot on our credit as a nation." I cannot unreservedly accept this view. No doubt, while he was making himself a name, *i. e.* while the worth of his researches to the world was still unproved, he was allowed to struggle on unaided; but I do not understand how this can be helped. The public could not reasonably be called on to give pecuniary assistance to untried investigators, nor would science be benefited by any such course. As soon as the sterling value of Parker's labours became apparent, public help was given him by the Royal Society

out of the public funds, and he was ultimately sufficiently provided for by the Civil List to enable him to retire. I do not say that a more liberal provision at both periods would not have been well bestowed, for Mr. Parker was a man so devoted to his work that he had no need of what John Hunter called "the stimulus of necessity" to prevent him from idleness; but others are framed in a less heroic mould, and I am sure that indiscriminate charity, veiled under the specious name of "encouragement of research," would neither be fair to the public nor beneficial to science. Mr. Parker, as I knew him, was a man of cheerful, independent disposition, who delighted in working in his own way, and who was proud, and justly proud, of having won his spurs, not without a hard conflict. I do not think he ever complained, or thought he had reason to complain, of others for not aiding in what he regarded as his own task. There are, we may reasonably hope, many such men in England, and I believe there are all the more because they are left to earn fame by scorning delights and living laborious days, without too much coddling from universities, institutions, or Government.

Another of our Honorary Fellows, *Sir E. Chadwick*, has passed away, after a long life spent in the service of his country and his kind, at the great age of ninety-one. Rarely, indeed, has it been given to a man to achieve so great a success in a single lifetime as has attended the labours of this great sanitary reformer.

When Mr. Chadwick first entered on his career, as the pupil, friend, and confidential agent of Jeremy Bentham, it is hardly an exaggeration to say that there was no such thing as sanitary science. Now it has taken so high a position, in consequence of its proved benefits in every department of public life, that only a few years since a prime minister could exclaim, "Sanitas sanitatum, omnia sanitas!" A well-informed writer in the 'Edinburgh Review' (Jan., 1891, p. 70) thus speaks of Sir E. Chadwick's early career:—"Time, and now at a ripe old age the grave, have softened the criticisms which assailed Mr. Chadwick

in the debates of 1854, when the conduct of the Board of Health was under review in the House of Commons. That he was high-handed, that he was intolerant of stupidity, that he was merciless to local corruption, that when he greatly desired an end he risked the loss of it by precipitancy in the means, that, like Frederick the Great's schoolmaster, he knew in some ways little of the world—this is one side of the shield. That he was a single-minded and devoted servant of the State, a pioneer of unremitting zeal in fields then unknown, a specialist who could administer, and above all an ardent lover of his kind—this is the other." "His first report," says one of his biographers in the public press, "on the sanitary condition of the labouring population of Great Britain, published in 1842, at once arrested public attention to a great blot in our social system; and though he had not received a medical education, the College of Surgeons paid him the compliment of seeking his assistance in its own special work, and of declaring that his report above mentioned was 'one of the most valuable contributions, beyond all question, to the noblest department of medical science, the art of preserving the health of the community at large.'" No less important was Mr. Chadwick's work on the subject of intra-mural interments, the cessation of which was due mainly—it would hardly be too much to say exclusively—to him; and this has brought about an improvement in the health of our great cities which alone would amply reward the efforts of a lifetime. Again, it would be difficult to exaggerate the service which he rendered to the State by his paper in 1858 "On the Application of Sanitary Science to the Protection of the Army in India." The inquiries to which this paper gave rise, the sanitary improvements which followed, and still more the knowledge of the subject thus given to army surgeons and commanding officers, has reduced the death-rate of our troops in India in thirty years to one third of its former scale,[1] and in so doing has proportionally improved the

[1] The Report of the Commissioners appointed to inquire into the sanitary

vigour and efficiency of the whole force. A greater boon could hardly have been conferred on the whole nation; in fact, he lived to be universally recognised as the father of sanitary science, and to obtain honour from his countrymen more precious than any titles or decorations which could be conferred on him by the Government. The latter, it must be confessed, were not too liberal. He was made a C.B. tolerably early in life, but it was not till the age of ninety that he received the honour of knighthood, an honour hardly worth having, one would think, so near the verge of the grave.

No one had rendered greater assistance to the medical profession in its main object—the preservation and prolongation of human life,—and no one more worthily occupied the honorable position of an Honorary Fellow of the Society.

Sir Wm. Hoffmeister, who died in July last at the age of seventy-three after a severe attack of bronchitis, was a trusted friend of the Queen, whom he had attended ever since Her Majesty and the Prince Consort took up their summer residence at Osborne in the year 1845. Dr. Hoffmeister was at that time a young man—having commenced practice at Cowes only four years previously, but the choice of Sir James Clark, then Her Majesty's chief medical adviser, was amply justified by the event, and the Queen was latterly in the habit of securing Sir William's services in Scotland also; while the fact that he went twice to Darmstadt to attend the Princess Alice, Grand Duchess of Hesse, in her confinements further

state of the Army in India, published in 1863, gives the annual average mortality per 1000 during the previous ten years as 51·2. The Twenty-fifth Annual Report of the Sanitary Commissioner with the Government of India for 1888 gives the same mortality as 14·84 per 1000, and this is explained to be a more unfavorable report than that of the previous year, in consequence of the statistics from Upper Burmah being included for the first time. The rate has in recent years been as low as 10·88. The total loss from both death and invaliding now is much less than the death-rate was then—38 against 51 per mille.

testified to the high value which the Queen placed on his medical skill and her personal esteem for him.

He was also distinguished for his warm interest in the Royal Medical Benevolent College, and was universally esteemed by the poor as well as by the great.

Dr. Wm. Webb, of Wirksworth in Derbyshire, had been a Fellow of the Society for more than a quarter of a century, and was a highly respected and well-known practitioner in that part of England—his father having been in practice at Barton-under-Needwood in the neighbouring county of Stafford. He served an apprenticeship to his father before proceeding to the School of Medicine at Birmingham, of which he was one of the most distinguished scholars. Having enlarged his medical studies by a visit to Paris, and served for two years as House Surgeon to the Stafford Infirmary, he established himself at Wirksworth, where he conducted a large practice and obtained a high position in society.

"Dr. Webb," says one who knew him, "was eminently earnest and thorough in everything that he undertook, whether it was in politics, in which he took an active interest, or in the promotion of the sanitary improvement of Wirksworth, which he sedulously advocated, or in pleading the cause of the Cottage Hospital, of which he was, from its foundation, the medical officer and active supporter. In all work for the public good he was ever a ready labourer, and great was the gloom cast over Wirksworth and the neighbourhood by the announcement of his death, for he was universally respected. An able operator, kind and sympathetic to his patients of whatever class, and with a wide knowledge of his profession in all its branches, Dr. Webb was an admirable specimen of the best type of general practitioner, and for many years enjoyed the confidence of a large circle of patients. During the latter years of a life that was too soon cut short, he was compelled by failing health to withdraw from general practice, and practised as a consultant in Derby and at Wirksworth.

"Dr. Webb graduated, when quite a young man, at the University of St. Andrews, and was a Fellow by examination of the College of Surgeons of London. He contributed various treatises of a practical nature to the literature of his profession, as well as to the local archæology of Wirksworth and its neighbourhood."[1]

His death was caused by tumour of the bladder, which was removed, but with only temporary success.

Dr. Matthews Duncan was so prominent and so vigorous a member of medical society in London that it seems strange to speak of him among those who have passed away. He was an excellent example of the virtues of the older school of practitioners—those whose preference is rather for the old than the new methods—rather for gradual improvement than for violent changes—rather for wise management than for showy operations. Yet Dr. Matthews Duncan himself bore no small part in the introduction of chloroform—the most striking and the most far-reaching change which has occurred during human experience, not in medicine only, but in all the numerous points at which our art touches on human feeling; a change which, by abolishing the fear of surgical operations, has more than anything else facilitated the introduction of those operative measures which our friend was so apt to discourage. It was as assistant to Sir J. Simpson, and while resident in his house, that Dr. Duncan took part in the after-supper experiment with the new agent, which has been so often described as to have become historical. It was most fortunate that this and other early experiences of anæsthesia passed over without any accidental fatality, which might have long delayed, though probably it could not have prevented, the adoption of the new method.

Dr. Matthews Duncan devoted himself from the first to the study and practice of obstetrics. He had attained a high position and great reputation in Edinburgh before the death of Simpson in 1870, and thought himself legitimately entitled to succeed to his chair in the University. The

[1] 'Brit. Med. Journ.,' Sept. 13th, 1890.

adverse decision of the electors loosened his hold on the Scotch metropolis, and on the retirement of Dr. Greenhalgh he willingly connected himself with the school of St. Bartholomew's Hospital, and commenced practice in London. Here his success was rapid and lasting. Besides a large private practice he found time to publish many important papers, and also his Lectures on Diseases of Women.

Dr. Matthews Duncan's membership of the Society necessarily coincided with the period of his life when his professional engagements prevented much other work. Hence we have only one paper from his hand in our Transactions, a very short but very valuable paper (vol. lxvii, 273) on Albuminuria with Parametritis, the work of a physician of ample experience and of the most accurate observation—a paper of which Dr. Champneys justly said that "the facts were new—the special connection of temporary albuminuria with parametritis was a valuable clinical fact which would relieve the physician of much anxiety in such cases."

He joined our Society too late to have as yet served in any office except that of referee of papers, in which his ample knowledge and large experience were invaluable.

Dr. Duncan is one whose name will not soon perish from the memory of his friends or from the records of medicine. He was a man of strong character, yet free from arrogance or contempt of others—justly self-confident, for he could not but know that his opinion was founded on extensive knowledge and wide observation. In some particulars he may perhaps have carried resistance to innovation too far; but those who know most of his branch of the profession will, I believe, be the most lenient to such a fault. He was an eminently sincere and honorable man, and he earned and possessed the respect of the whole profession, who felt as a common loss the closure of a career from which they still hoped for much more of valuable work.

Professor Bigelow, who died on October 30th, 1890, aged

seventy-two, was an Honorary Fellow of this Society, and was unquestionably the best known of American surgeons in this country. He was a member of a family which could trace their descent from the original English source, and which had been long known and respected in Massachusetts, though originally settled, it seems, in the neighbouring State of New York. Professor Bigelow was compelled when young to travel in Europe for his health, being then suspected of a tendency to phthisis (a suspicion confirmed many years afterwards by post-mortem examination), and after his return and his graduation in medicine he again spent three years in medical studies, chiefly in France. He was one of the first to promote the introduction of anæsthetics. But his chief claim to fame in surgery—and it is a claim which is absolutely indisputable—rests on two works; that on Dislocation of the Hip and its Manipulative Reduction, and that on the Removal of Stone from the Bladder by the Lithotrite at a Single Sitting. In both these important particulars Professor Bigelow succeeded in establishing the practice which he recommended. It is true that the somewhat common opinion that Bigelow *introduced* the method of manipulative reduction of hip dislocation is entirely erroneous. He himself points out (op. cit., p. 28) that the principle was known to Hippocrates; and it is indisputably true that an Italian surgeon, Fabbri, had shortly before dealt fully and very satisfactorily with the matter in an elaborate treatise, which had not, however, I believe, come under Bigelow's notice. But all this in no way detracts from the honour due to the American surgeon, who worked out the subject for himself, and provided the surgical world with a very probable theory and with a series of manipulative directions which have amply satisfied the test of application to practice in numberless instances. Fabbri's work, having never been translated, remains almost unknown; while Bigelow's name has become, and will probably always remain universally associated with the anatomy of the hip-joint and the method of reduction of its dislocation.

His proposals for what he called "litholapaxy," again,

have equally withstood the rigorous test of practical experience. I will not spend time in discussing how much of originality there is in the principle of Bigelow's method, or whether it is merely an inevitable consequence from the fact of the dilatability of the male urethra demonstrated by Otis. The fact remains that it was sufficiently novel to arouse strenuous opposition from many surgeons of ample knowledge and experience, and that it has now passed into common use, with great advantage both to the rate of mortality, and to the rapidity and painlessness of recovery. That exaggerated expectations may have been formed of it, and that it may have been used in cases where lithotomy would have been less dangerous, is probably true; but similar drawbacks have attended every other advance in surgery.

Professor Bigelow retired some years before his death, not I believe as the result of failing health, but to follow country pursuits which were pleasing to him, and to enjoy his well-earned leisure. But his health soon gave way, and he died at an age when many surgeons are still able to give their colleagues the benefit of their ripe experience and practical sagacity in consultation. His name will always be cherished in America as one worthy to rank among the great surgeons of that country, and will never be dissociated in the surgical literature of the world from the two subjects which he had made especially his own.

Dr. Lewis Shapter, of Exeter, died at the early age of forty-two. He joined our Society at an early period of his career in 1873, the same year in which he was elected Physician to the Devon and Exeter Hospital. He was a man of liberal education, obtained at Westminster and Cambridge—an eloquent speaker and able debater. "As a Westminster scholar," says a friend of his, "he made full use of his privilege of listening to the debates in the House of Commons. Its laws of debate were at his fingers' ends, and thus he made an excellent chairman at public meetings." His mother was descended from Dr. Blackall, and more remotely from a former Bishop of

Exeter of that name. Dr. Shapter's wife unfortunately predeceased him, leaving an orphan family. His father, who was also Physician to the same hospital, survives at an advanced age. Illness compelled Dr. Lewis Shapter to resign the post he had filled with great success at the hospital, and he was elected Consulting Physician and Honorary Governor on October 23rd, 1890, but only survived for a few weeks. The same friend thus summarises Dr. Shapter's career:—"Dr. Shapter's scholarly address to the members of the South-Western Branch of the British Medical profession in his year of office as president will long be remembered by those who were privileged to hear it. Amongst his writings, which were numerous, the most noticeable are—The Thurston Speech, delivered before his college at Cambridge; a paper on the Medicinal and Dietetic uses of Citrate of Caffeine, and one on Functional Athetosis and Inco-ordination of Movement. Dr. Shapter was a Fellow of the Royal Medical and Chirurgical Society of London, and a member of several other scientific societies. His talents as a lecturer were well known to members of the Literary Society, of which he was one of the vice-presidents, and before whom he had given many interesting lectures on subjects of general and scientific interest. The gap caused by his enforced retirement and premature death is one that will not readily be filled, the combination in his person of scholarly attainments and scientific erudition with business-like habits being rare."

Mr. John Marshall died somewhat suddenly at the house to which he had recently moved in Chelsea on New Year's Day of the present year, at the age of seventy-two. No name deserves greater honour in our Society, for he had served it long and zealously, and in especial had presided over it for two years with an energy and success that had strikingly increased its efficiency and its reputation.

Mr. Marshall's career as an anatomist began under Ellis, as a physiologist under Sharpey, and as a surgeon under

Liston; and, as his natural gifts were great, it is no wonder that he attained eminence in all these branches of the profession. In anatomy he will always be remembered by his work on the Development of the Great Veins; and to the studies of the anatomists of his own day he contributed most useful assistance by his labours as demonstrator of anatomy in the school of University College in succession to Professor Ellis, and by his services as co-editor of Quain's Anatomy. In physiology he was well known by his 'Outlines of Physiology,' published in 1867, which was long one of the most popular text-books for advanced students. In surgery his chief original work was the introduction of the electric cautery (of which I shall speak more in noticing his contributions to our Transactions), and his proposal for the removal of varicose veins by the knife. The latter suggestion excited much difference of opinion, and at first rather warm opposition.[1] It was not introduced as being a novelty, having in fact been practised by Celsus, but it had fallen into entire disuse, and was indeed too dangerous to be justifiable, previous to the great improvement in the treatment of wounds which has followed the introduction of the antiseptic method—a method of which Mr. Marshall was an enthusiastic and ardent follower. All opposition to Mr. Marshall's views on the treatment of varicose veins has now ceased, and excision has taken its place as a recognised method of cure in the more severe cases of varix, whether in the extremities or the spermatic cord. On the subject of nerve-stretching also, and on that of the removal of portions of the lower bowel in the extirpation of tumours (colectomy), Mr. Marshall published excellent papers.

Another department in which Mr. Marshall was eminent above all his contemporaries was in the popular exposition of anatomy and physiology. Mr. Marshall held for four years the office of Fullerian Professor of Physiology at the Royal Institution, and he was the first to lecture on

[1] See the original paper in 'Lancet,' 1875, vol. i.

this subject to female students at University College. At Marlborough House also, and afterwards at South Kensington, he gave lectures on anatomy as applied to art —courses which were continued over a period of twenty years, and which showed the practicability of treating the subject before mixed classes of men and women. On the death of Mr. Partridge he succeeded to the office of Professor of Anatomy at the Royal Academy, and here, also, the lady students were admitted to most of the lectures and demonstrations. These lectures of Mr. Marshall were eminently successful, and form the basis of a work on the subject which had reached its third edition just before his death.

Mr. Marshall's contributions to our Transactions were three in number. The first is one of great interest and great surgical importance. It is contained in vol. xxxiv, and was written just before he became a Fellow. It describes the invention of the electric cautery, and records the first case of its application, for the cure of a fistulous opening in the cheek, with complete success. The paper is an excellent example of the care and thoroughness of Mr. Marshall's research, and his foresight in estimating the therapeutical value of his invention. Even at the present day, and after forty years' experience of the electric cautery, we could hardly give a more complete account of its applications than Mr. Marshall gave in 1851 in these words :

"For all cases in which cauterisation may be advisable, and in which neither the common heated wire nor button is available, the electric cautery will, I feel sure, be employed with advantage ; as, for instance, in obstinate fistulæ leading into the mouth, or communicating with the trachea, in intractable perineo-urethral or recto-urethral fistulæ, for cauterising the edges of vesico-vaginal fistulæ, or the internal surfaces of long sinuses, for arresting hæmorrhage in certain parts otherwise difficult of access, for uniformly cauterising deep poisoned wounds, and in other conceivable instances. But during the progress of

my experiments another and very different application of the electric cautery suggested itself, viz. that of employing the incandescent wire in the section of soft parts. In the experiments on dead animal tissues I had found that a considerable thickness of muscular substance could be cut through by the heated wire in a few seconds; and I had further ascertained in the living animal that no hæmorrhage occurred from the division, by the hot wire, of veins as large as a crowquill, or of arteries a very little smaller.

"It seemed probable, therefore, that it would be simple and safe to apply this method for the removal of redundant vascular parts, instead of the knife, scissors, or ligature; as, for example, in hæmorrhoids, external or internal; polypi of the uterus, and certain forms of erectile tumours or any other soft pedunculated growths. For extirpation of portions of the tongue or of the os uteri it also appeared likely to be useful. Moreover, in certain peculiar cases, where simple division of the tissues alone was required, it seemed worthy of trial, as in the various kinds of rectal fistulæ, or in fissure of the anus. As a means of obliterating varicose veins it might also prove safe and efficacious. Lastly, in the case of persons having a strong hæmorrhagic tendency, this mode of dividing the soft parts appeared to offer great advantages."[1]

The other two papers are (vol. xxxv) an interesting case of chronic obstruction from pins swallowed, probably accidentally, and lodged in the stomach and duodenum; and (vol. lxvi) a short history of a case in which there had probably been laceration of the kidney, followed by a suppurating hæmatoma, cured by antiseptic drainage. This paper was communicated during his Presidency.

Mr. Marshall started on his surgical career as Liston's assistant in private and assistant surgeon to University College Hospital, and after filling all the active surgical offices there he became Consulting Surgeon in the year 1884, after thirty-three years' service.

Much of his active life was spent in the service of our

[1] Op. cit., p. 229.

public institutions, and much of his great reputation was derived from this part of his work. He gained the Fellowship of the Royal Society early, and twice served on the Council. Of his services to our own Society I have already spoken, however imperfectly. At the College of Surgeons there were few Councillors whose opinion was more respected, or whose capacities for business were more highly valued than his. I was one of his colleagues on the Council for many years. I was not always able to assent to his views, and especially regretted the unnatural separation made between the sciences of anatomy and physiology in the examinations, mainly owing to Mr. Marshall's strenuous advocacy, though his own example was a forcible proof that a surgeon could cultivate both with distinguished success. But I was fully alive to his great capacity as an administrator, his ardent zeal for progress, and the courtesy and kindness which he showed even in opposition; and I joined with all his fellow-Councillors in their high estimate of the benefits which he procured for the College in the promotion of the conjoint scheme, the introduction of the examination in elementary anatomy and physiology, the reduction of the admission fee, and many other particulars.

He was chosen, many years before his death, to represent the College of Surgeons on the General Medical Council, and at the time of his decease held the office of President of that body, in succession to Sir Henry Acland. He had not held this high office long before his death, but it is impossible to doubt that, had he been longer spared, so good a man of business and so zealous a practical reformer would have marked his career as President by some valuable public service.

I hardly like, in a public notice of this kind, to speak of a man's private character, and will only allow myself here to say that much of the great success which Mr. Marshall obtained in his public career was due to the kindness, affability, and gentlemanliness (in the highest sense of that much-abused term) which endeared him to

his private friends, and which will cause him to be regretted so long as any of them survive.

Mr. Bellamy, late Surgeon and Lecturer on Surgery at Charing Cross Hospital, died suddenly from acute inflammation of the lungs on January 5th of the present year, at the early age of forty-eight, and while apparently in the prime of vigorous manhood. His death was thus closely successive to Mr. Marshall's, and there was much similarity in their artistic tastes and their capacity for popular exposition. Mr. Bellamy succeeded Mr. Marshall as Professor of Artistic Anatomy and Examiner in the Art Schools at South Kensington, and might very probably have been his successor at the Royal Academy. He was a facile draughtsman, a good anatomist, an ardent surgeon, and a dexterous operator. He died at too early an age to have attained full professional success, but everything seemed to promise it, and his premature death probably deprived the surgical profession of one who would have added much to the progress of our art and done much worthy service to humanity.

Mr. Bellamy had served this Society as a member of Council, a Referee of Papers, and a member of the Library Committee. His only contribution to our Transactions is an interesting account of an operation by which he removed a tumour, probably sarcomatous, from among the cords of the brachial plexus, with success for the time the patient survived, but she died from another cause about half a year afterwards. The discussion of this paper was made more interesting and valuable by the fact that Mr. Bryant was able to relate an almost precisely similar case in his own practice followed by permanent success.

Amongst the many deaths from acute pneumonia which have occurred this winter was that of *Dr. G. Gulliver*, of St. Thomas's Hospital, the son of the well-known surgeon and naturalist. Dr. Gulliver inherited his father's love for natural science and zoology. He was a member of the University of Oxford, and while there was a pupil

of Professor Rolleston, on whose recommendation in 1871, when only twenty years of age, he accompanied the expedition to observe the transit of Venus in the capacity of naturalist, and on his return received the thanks of the Royal Society for his report on the zoology of the island of Rodriguez in the Indian Ocean. Four years elapsed before hecommenced his medical studies at St. Thomas's, where he became Assistant Physician and Lecturer on Comparative Anatomy in succession to Mr. Stewart, now Curator at the College of Surgeons. In 1883, when cholera was epidemic in Egypt, Dr. Gulliver was one of the physicians despatched thither by the Foreign Office, and besides the great services which he rendered there he read a paper on his return before the Epidemiological Society, on the Etiology and Pathology of Cholera. This paper, which is spoken of as valuable, and some good work in the Pathological Transactions and in the St. Thomas's Hospital Reports, are all the medical treatises that Dr. Gulliver completed. He had stored up the experience he acquired as Physician to the London Fever Hospital for a work on fevers, which he had only just begun when his sudden and fatal illness attacked him. He had been only for five years a Fellow of this Society, and had not contributed to our Transactions.

Dr. T. Graham Balfour, who died in the month of January in his seventy-eighth year, was one of the oldest members of our Society, the date of his election being 1839. He was a member of a good Scotch family, his great-grandfather having been a Professor in the University of Edinburgh. He was connected also with the literary world of Edinburgh, being an intimate friend of Dr. John Brown and a relation of Mr. Robert Louis Stevenson, and having studied at the University when it numbered Wilson, Alison, Chalmers, and Christison amonst its professors. He took the degree of M.D. there in 1834, and in 1836 joined the Medical Service of the Army. He was at first employed on the statistical work of the Army in company with Sir A. Tulloch—digesting

returns which had been accumulating since the date of Waterloo. Afterwards he served with one of the battalions of the Guards for some years; but in 1847 resumed statistical work along with Colonel Tulloch, and after the Crimean War he was joined with the same officer on the commission which investigated the causes of the breakdown of our Military Medical Service at the beginning of that campaign. The report of this Committee was a main cause of the thorough re-organisation of the service, and it is believed that still better results would have followed had its recommendations been more strictly adopted. In 1859 Dr. Balfour was made the head of the newly organised statistical branch of the Army Medical Department, and for fourteen years "he continued to edit the statistical reports of the Army, which are among the most precious data not only of the military administrator, but of epidemiologists in general."[1] He continued in the Army Service till 1876, when he retired after forty years' service with the rank of Surgeon-General, and in 1887 was appointed Honorary Physician to the Queen. He was elected a Fellow of the Royal Society in 1859. His work in the Army was not confined to statistics, for he was in charge of Netley Hospital for a time, and afterwards of the Medical Department at Gibraltar. He gave an active support to the Statistical Society, of which he was for a time President, and he gave his services to the public at the close of his life on the Metropolitan Asylums District Board. He also took part in the Committee which introduced the system of the Contagious Diseases Acts, and was one of the great majority of medical men who lamented the abandonment of the only system which seems likely to put a check on the ravages of syphilis and to mitigate the horrors of prostitution.[2]

In this Society Dr. Balfour felt a warm interest, and he served it in many capacities—on the Library Committee in 1849, on the Council in 1852–3, as Vice-President in

[1] 'Brit. Med. Journ.,' Jan. 24th, 1891.

[2] See a letter from Dr. Wilks, 'Lancet,' Jan. 31st, 1891.

1860–1, and as Treasurer in 1872. Dr. Graham Balfour's contributions to the Transactions were two in number —viz. (1) in vol. xxxv an interesting and convincing demonstration of the benefits of vaccination, derived from the experience of the army and navy in various climates, and that of the Royal Military Asylum ; and (2) an investigation of the value of the spirometer in forecasting the liability to pulmonary disease, as shown by the subsequent history, during a given period of time, of a large number of recruits whose "vital capacity" had been thus ascertained. This paper is an interesting example of the valuable results obtained for medical science by the old system of long service and accurate medical histories continued over a series of years.

Dr. Balfour was an admirable example of his nation and of his branch of the profession—with all the sagacity, sound sense, and devotion to duty of a Scot of the old school, and with the wide knowledge of the world and intelligent patriotism which distinguish the Military Medical Service.

The last on our roll of deaths is *Dr. Morris Tonge,* of Harrow, a man who in his earlier days was especially ardent in his devotion to pathology, and was one of the best known members of the Pathological Society, but who was afterwards absorbed in the cares of an extensive practice, though he still occasionally found time to show that his earlier interest in the scientific part of our profession was unextinguished. Dr. Tonge died only a few days ago, at the comparatively early age of fifty-four, a great loss to his profession and to the wide circle of his friends.

We will now pass from the contemplation of the losses which death has caused us during the past year to consider the present condition of the Society and its future prospects.

The year during which I have had the great honour of occupying this Chair has been one of much interest and

of the greatest importance to the future of the Society, since it was the first year in which we have had complete occupation of our new premises, and has been the period during which all the complicated operations necessitated by the great change in the Society's position and relations have been finished, or at least brought to something like a practical termination.

These operations will be found described with sufficient fulness in the Council's Report, and I will not, therefore, consume the time of the Fellows by recapitulating what is in the hands of all of them already. But I may be permitted to put before you my view of the net result thereby obtained, especially as two opposite opinions are entertained of the position of our Society, even by well-instructed persons. Some believe that the speculation into which we entered when we bought our present house and undertook such large and expensive additions to it has failed if regarded as a commercial undertaking, and that the Society is not likely to make both ends meet : and this view of the situation was brought before the late special meeting with much ability by several deservedly respected Fellows. On the contrary, there are those who say that the terms on which we have admitted other societies as tenants are exorbitant, and have resulted in putting this Society in possession of its premises rent free, and with a surplus in the near future, for which there will be no legitimate use. I do not share either of these extreme views. It is perfectly true that when our interest in the Berners Street premises expires, *i. e.* at the end of the year 1904, £450 of income (or to speak more accurately, allowing for the ground rent we pay, £436 7*s.*) a year will cease, and if our ordinary income shall not have at all increased, and if the repayment of bonds shall not have amounted to £10,900, the Society will not be in as good a financial position then as it is now. Nor do I expect that we shall have diminished the debt in fourteen years by so large a sum as that.[1] But I do expect that the

[1] If we put by £1,700 a year for the charges of the debt, as contemplated

ordinary income will have risen quite sufficiently to make good the deficit. The income of the Society has been gradually rising for a considerable time—in fact, as long as I have investigated the question ; and it will, and must, rise so long as a number of contributing Fellows join in each year equal to those who died or resigned in the previous year, for the plain reason that the majority of the Fellows who die have compounded and ceased to contribute,[1] while all those who join do at any rate contribute in that year. But, as a matter of fact, the number of Fellows contributing has been steadily rising for many years,[2] and I see no reason why this increase should stop, now that the Society has got into a better position, and shows all the signs of more vigorous life. So that I regard our pecuniary prospects without any apprehension. Yet I cannot see how the Building Committee could have afforded, with due regard to the stability of the Society for which they were trustees, to let the premises at a cheaper rate, or to do anything tending to the diminution of the Society's income. So strongly did we, the members of that committee, feel this, that we decided, though with regret, to part with the first-floor room, which had been at first used as the council-room, in order to make the more sure of being able to meet the obligations of the Society to its bondholders. It is true that the income from rents is large, and if the letting of the two rooms which are available for day and evening meetings fulfils our expectations, we may realise a surplus considerably larger than the modest sum (£174) which we estimate for the present year as available for contingencies, after the provision of £1,700 for the charges of the debt. If that should turn out to be the case, the surplus would, of course,

in the Report laid before the Fellows at the late Special General Meeting, the repayment of debt would have been about £5,000 by the end of 1904.

[1] Of the 19 Fellows who died during the past twelve months only six were still subscribing. To this number must be added one who resigned.

[2] The number rose very gradually in the ten years 1881—1890 inclusive, from 292 to 368, and the income from fees, &c. (including sale of Transactions), from £1,264 to £1,638.

be available for the further reduction of debt, and we might even have extinguished enough of our bonds by the end of 1904 to balance the loss of the Berners Street rent. But I think anyone would admit that if we neglect any such hypothetical increase of income, this estimated balance is by no means too large to provide for the many contingencies which may swell our expenditure.

There are, however, various matters in connection with finance which deserve, and which I am glad to see are receiving, the attention of the Council and of the Fellows. Our Bye-laws are based on a state of things which has long passed away. The city in which this Society was founded in the year 1806 was as different a place from the London of our day as the old house, in which we are located, then was from the extensive premises into which we have now transformed it. London then contained no cabs, no omnibuses, no railways. A mile from this spot was open country, seven miles from the post office was a formidable journey, hardly to be risked in the dark on any terms, and certainly not without arms. A physician would not drive his coach "off the stones" without an extra fee. The postal service was slow, infrequent, and very dear. In fact, the town was comparatively small, and was sharply marked off from, and had only rare intercourse with, the country. Modern changes have tended to alter the relations between residents and non-residents, and the Bye-laws which define and separate these classes, and which prescribe their respective privileges, are obviously in need of reconsideration. And such reconsideration was actually commenced by the Council, but the pressure of other business has prevented any final conclusion being reached on the subject. All I need say is that the limit of non-residence seems (to me at least) much too near, considering the facilities we enjoy for travelling between the centre and the suburbs; but that, on the other hand, nothing ought to be done to infringe on the privileges of any of the present Fellows. I trust, however, that this matter will be ready for the con-

sideration of a general meeting at some reasonably early period.

Another question was mooted at the Special Meeting on February 18th, and has also been discussed by the financial authorities of the Council; but, like the limit of non-residence, has been hitherto deferred from the pressure of more urgent business—I mean the equitableness of the present scale of composition fees. We believe that, considering the great diminution in the rate of Government interest since the time at which the present scale was fixed, that scale must now act prejudicially to the funds of the Society. On this subject the Council will no doubt before long take skilled advice and report the result to the Fellows.

So much for financial matters. Let us now turn to the general affairs of the Society.

This Society may be looked on in various ways. It is, in the first place, an association of medical men, whose character and position are guaranteed by the ballot through which each member has to pass, and which is by no means an idle ceremony. The great majority of the leading physicians and surgeons of London, and many of those in the country, belong to our body. It is well that we should occasionally ask ourselves whether our corporate action is on a level with our position and with the personal qualifications of our members. Is there sufficient feeling of fellowship in this great and ancient Society? It has rendered great services to the medical sciences, it is true, and we may well be proud of a series of Transactions which is, we believe, unequalled in value by any serial medical publication in the world. But the founders of our Society evidently contemplated some personal intercourse among its members; for besides the reading of letters, reports, and other papers on Medicine and Surgery, &c., we are directed by Bye-law, Ch. xvii, § 3, to "converse upon professional subjects." The latter object has been overshadowed, and in practice thrust aside by the very excellence of our Transactions, which has led to

so constant a supply of papers of sterling value that no time remains at the ordinary meetings for any conversational friendly intercourse. Would it not be a good innovation, and one strictly in the spirit of the above Bye-law, if some meetings of a less formal character were held occasionally—such as a house-dinner or a conversazione once or twice in the session, at which the Fellows could learn to know each other better, and to converse in a more unrestricted manner than is possible in the debate which follows the reading of a scientific paper?

I leave the suggestion for what it is worth in your hands, satisfied that if it commends itself to the Society the Council will soon find means to act on it.

Another and a graver question forces itself on our minds in considering this aspect of our Society. This is, I will not say *whether*, but rather *when*, it will be desirable to take up again the subject which was mooted some thirty years ago, first in 1860 under Mr. Skey's presidency, and resumed in 1861, when Dr. B. G. Babington was President, and our late President, Sir E. Sieveking, was Secretary, along with the lamented Mr. C. H. Moore. That subject I need hardly remind you was the amalgamation of the various societies then existing for the study and promotion of the different branches of medicine into one great institution, of which the Royal Medical and Chirurgical Society would be the stem, and the Pathological, Epidemiological, and Obstetrical Societies the branches.

Since that day the Clinical Society has come into existence, and a second obstetrical society has been founded under the name of the Gynæcological.

All these societies are housed with us with the exception of the Epidemiological.

That the effort at amalgamation then made was not successful seems to me, on a reperusal of the journals of that day, to have been less the result of opposition in this Society than of the dissent of some of the others which were to have been incorporated. There was, however, a

considerable difference of opinion amongst our own Fellows also, though the proposition which Mr. Charles Hawkins made, accepting and embodying the principle of amalgamation, was carried after a prolonged debate in a tolerably full meeting of the Society by a majority of thirty-eight to twenty-three. Even now many of our most respected and most enlightened Fellows no doubt are inclined to the opinion not obscurely intimated by my respected predecessor, Sir E. Sieveking, in the first Address which he delivered in this house,[1] viz. that the "objects that an Academy of Medicine would have in view would be, perhaps, even more energetically and beneficially realised by the co-operation of societies that are now distinct in their objects, their means, and their government." And it is useless to hide from ourselves the difficulties which beset the scheme, not only pecuniary (though these are considerable), but those arising out of the legitimate, nay, even praiseworthy, jealousy of the minor societies in defence of their independence and freedom of action. Yet I cannot think these difficulties insurmountable. With the valuable property which our Society has acquired, and which will increase every year in value (unless all those who have been concerned in your affairs during the last two years are grievously mistaken), the question of money need not be regarded as insoluble; and with regard to the freedom of action and the dignity of the minor societies I need only say that we are almost all of us members, and warmly attached members, of one or more of these same societies, so that the great majority of our Fellows are as much interested in safeguarding the rights of those societies as are those who are their exclusive members.

I do not say that the time is ripe for the practical discussion of this matter; in fact, my own opinion would rather incline to defer it till a few years' experience has tested the financial security and the scientific progress of this Society in its new situation; but at no long period the question will inevitably be rediscussed, and I for my

[1] Oct. 22nd, 1889, Transactions, vol. lxxiii, p. xcvi.

part am willing to enter on it now, if there is any general consensus of feeling in the Society and the profession to that effect.

Meanwhile, if we shrink from undertaking the general question now, cannot we do something even now to render the development of a general institute of medicine out of the present Society easier? Is it creditable to us in London that the Epidemiological Society should be the only association, and that a merely private one, and one not strictly medical, for the important public duty of studying the laws of public health? Is it creditable to us that the sciences which are the foundation of rational medicine—Anatomy, Physiology, Chemistry—are getting more and more separated from practical teaching, more and more matters of "cram" to be "got up for exams." —if I may be pardoned for using the hideous slang of the day—and that there is no provision in this great Society for their scientific pursuit? The two English Honorary Fellows who died during the past year, Mr. Parker and Sir E. Chadwick, were eminent examples, the former of the charm which the study of scientific anatomy can lend to a life passed in a hard struggle for daily bread, the latter of the incalculable service to humanity which the devotion of a long life to sanitary science, even without a knowledge of medicine, can render. Our Society contains men not less gifted nor less qualified to promote such studies than those who have passed away. Can we not then do something to carry out more fully the object specified on the first leaf of our Charter as the aim of our foundation, "the cultivation and promotion" not only "of Physic and Surgery," but also "of the branches of science connected with them"? In the former we have done much, why should we do nothing in the latter?

Perhaps I am going too far in saying that we do nothing in the directions suggested. We are now awaiting the results of an investigation undertaken by one of our "scientific committees" on the subject of the Baths and Health Resorts of this kingdom, a question of great

importance in practical medicine, and of much interest also in regard to the public health. Another scientific committee has been appointed to reconsider the question of the treatment of suspended animation from drowning, which again bears on public medicine. These are steps in the right direction, but I cannot help thinking that, in the absence of any action on the part of the Colleges, our Society might render important aid to the Government and the Legislature in the investigation of the many important problems of sanitary science which yet remain unsettled.

So, again, with regard to anatomy and physiology. Valuable papers no doubt exist on such subjects in our Transactions, but they are few in number, and do not tend to become more numerous, and they are almost exclusively directed towards practice. A Physiological and an Anatomical Society exist and do good work. The College of Surgeons has recently been enlarged, and every facility has been provided there for carrying out investigations of all kinds. Our Society contains as Fellows most of the prominent members of all three bodies. Could we not find means, through our scientific committees, for bringing them into working order with each other, and organising such researches as appear to promise the best and most immediate results?

And in chemistry and pharmacology ought we to remain content without any action on the part of our own Society?

As a society for promoting the study of the great sciences of medicine and surgery, I think we have no reason to shrink from criticism, nor do I believe that the interest of our debates or the value of our Transactions shows any decline, or any tendency to decline. In looking through the records of our old debates on the subject of the Amalgamation of the Societies, I was amused to see the solemn warnings which were then addressed to us as to the decay of this Society, and the "senility" which was then attributed to us. Whether this might or might

not have been true thirty years ago it is hardly worth while now to discuss. It is, at any rate, not true now. We advance at a rate quite equal to that of the younger societies in spite of our larger subscription, and we can look forward with great confidence to an increased rate of progress in proportion to the increased advantages which our membership will offer.

It would hardly be decent in the President of a Society like this to undertake a criticism of the contributions of the Fellows, and if this could be permitted to any President, I am conscious that I am myself unequal to the task; but I may be allowed to say that some of the discussions which have occurred during the year have been on subjects of the deepest pathological interest and the highest practical importance, that the manner in which they were discussed was worthy of their intrinsic interest, and that the audiences which listened and took part in the discussion were large, and consisted of men whose eminence lent additional authority to their opinions. Many also of these papers were contributed by Senior Fellows of high position both in this Society and in the world outside. It is natural enough, perhaps, that not only our critics, but the officers of a Society like this, should regret that our older members do not take part more frequently in our proceedings and discussions. And it would no doubt be an incalculable advantage if they could find it possible to do so. But the limited experience of my own tenure of office encourages me to hope that the Senior Fellows are now disposed to lend more active assistance in the future than they have done in recent years, and in any case we must make much allowance for men who have borne the labour and heat of many a day of toil and anxiety, and who may well feel both desirous of, and entitled to, repose.

Another and to my mind a most important feature of our publications, and one which I fear is too little appreciated, is our Proceedings. I am afraid that this valuable series is looked upon by many of our Fellows chiefly

in the light of a grave for the decent burial of the abstracts of papers not accepted for the Transactions ; but in reality, through the energy of our resident librarian and the willing co-operation of the Fellows taking part in our discussions, the Proceedings have become a most valuable—I might say indispensable—adjunct to the Transactions. Any physician or surgeon working at any subject will at once appreciate the difference in the value of a paper in our Transactions, which on the old system merely gives the opinion and experience of the author, and one in which, as is now the case, that opinion is modified and that experience amplified by the comments of an audience of men such as those who attend our meetings, and reported in such a manner that the reader can be sure of having the speaker's words and opinions revised by himself and stated in plain and intelligible language.

I was profoundly impressed on reading the interesting paper by the late Mr. Gregory Forbes in the Transactions for 1849, on a case of lodgment of a foreign body in the bronchus, with the loss that we have sustained by the non-preservation of our debates, when I remember the interesting and instructive discussion which followed on a similar case (only treated with success instead of being allowed to end fatally) by Dr. Cheadle and Mr. T. Smith, recorded in vol. lxxi. The records of such debates in old times would have given us precious assistance in studying the history of medical and surgical opinion. Everyone who desires to make full use of our Transactions should keep the series of Proceedings with the same care as he does the Transactions themselves.

Another and a most important feature of this Society is its Library. It is true that at the College of Surgeons there is a medical library possibly more extensive and certainly more magnificently housed than our own. But that is only a reading library, while ours is a circulating one also—a difference of immense importance to men so busily employed in the daytime as our Fellows are. It is, therefore, one of the most urgent duties of the Society

to maintain, and, if possible, extend, the size and efficiency of their library. We cannot deny that during this transition period, when the Society had neither funds to purchase books, nor premises to receive them, nor any conveniences for their perusal, nor any staff with leisure to superintend their distribution, the library was of necessity checked in its development. The accounts for 1889 include only £40 for books and binding. Those for 1890 are somewhat better, and the same item stands at £139. But though this shows some improvement, it is far below the sum required to keep a great library like this supplied with a sufficient amount of new material. It is, however, satisfactory to be assured by the librarians that "all periodical series and works in continuation have been kept up to date; that all works specially recommended by Fellows have, as far as possible, been procured, and that the binding has not been allowed to fall into arrear." In framing the estimate for 1891, contained in the report laid before the late special meeting (February 18th), we have put down the probable expense of the library in the present year at £300. Some of the Fellows suggested at that meeting that the sum was inadequate; and I quite agree with them that a larger expenditure might be fairly incurred in future years, if the funds of the Society admit it, or even in the current year if we see that our receipts are exceeding our anticipations. Meanwhile we thought that an expenditure more than double that of the past year would suffice for the absolute needs of the day.

Another matter connected with the library is the absolute separation made by the bye-laws between the resident and non-resident Fellows in regard to its use. No non-resident Fellow can make any use of the library (except as a reading-room when in town) on any other terms than by payment of the subscription of a resident. If the Fellows assembled to-day accept the recommendation of the Council this will be so far altered as to give non-residents the privilege of taking out one volume at a

time, for a subscription of one guinea. Since the old bye-law was framed the Parcel Post and railway facilities have so altered the circumstances of life in the country, that country residents can take out books, or have them sent down to them to an extent formerly impossible. It is hoped, therefore, that the Society will sanction an experiment which, at any rate, can do no harm, and which if successful will be both convenient to country Fellows and beneficial to the finances of the Society.

I trust to the indulgence of the Fellows if I have ventured beyond my province in suggesting matters for their consideration which have occurred to me as calculated to increase the efficiency of the working of our Society. It must be recollected that, though the Council are charged with the management of our affairs, yet the spirit in which they are managed, and their general scope and aim, depend on the action and opinions of the Fellows at large, and that as no change which the Council might recommend could become valid without the consent of the general body, so the Council would not be well advised to embark on the serious consideration of any such change without good reason, at any rate, for believing that it would be agreeable to the Fellows. Our Annual Meetings, and the address of the President, seem to me a fitting opportunity for introducing general questions like these—which might be further ventilated and discussed at such social gatherings as I have hinted at—and so the Council be encouraged to embark on new plans of action with confidence of support from the Fellows.

Time will show whether the opinion which I have expressed, and which is, I believe, entertained by the great majority at least of the Council, is correct, viz. that the Society is entering on a period of increased resources, increased numbers, and extended means of action of all kinds. Nor is any long period necessary. The experience of the current year will probably enable us to form a reliable judgment on this subject. But you will, I am

sure, all agree with me that if our means do increase, our responsibilities will have increased in the same ratio. And I feel certain that we may with confidence leave it in the hands of the present race of Fellows to see that, in that case, the brilliant services which the Royal Medical and Chirurgical Society has rendered in past times to the profession and the public shall be eclipsed by its performances in the future.

Nothing remains for me but to render my best thanks to you for the distinguished position in which your suffrages have placed me, and for the support with which you have honoured me during my year of office.

SEVEN CASES

OF

EXCISION OF THE HIP COMBINED WITH THE HOT-WATER FLUSHING METHOD;

PRIMARY UNION IN SIX.

BY

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Received May 12th—Read October 28th, 1890.

More than a year ago I had the honour to present to this Society a paper on "Primary Union after Excision of the Hip for Tubercular Disease," which was read on December 11th, 1888. The aim of that paper was to show that certain conclusions as to the nature of tubercle of joints which I had put forward in my Hunterian Lectures, if correct, ought greatly to modify the treatment of this disease hitherto adopted; further, that cases of advanced hip disease, if treated by excision on the principles there laid down, would give better results than any we had ever yet seen from operation, and probably also better remote results. Finally, I desired to show that after this operation, even for advanced disease, primary union of the whole wound ought to be the rule if done at the proper moment.

The case which formed the text of that communication was one of advanced hip disease, with destruction of the

head of the femur. I had selected it as a test of the correctness of the conclusions put forward. As was anticipated, the whole excision wound had healed by first intention throughout, without a drop of suppuration, and has remained soundly closed ever since.

Since writing the above this boy has been brought to me. He walked well, and without pain, ever since he was shown at this Society in December, 1888. He had recently had scarlet fever, and looked a little debilitated. Though he had no pain or trouble in walking the mother was a little uneasy on account of a small swelling which she had noticed a week before under the old scar, which was still perfectly sound and white. This I examined, and found quite painless, but distinctly fluid. I therefore admitted him into hospital, and cut into the swelling just external to the old scar. It proved to contain a few drachms of thin glairy fluid, and ran backwards in a thin track to the trochanter. There was no disease of bone to be detected. Apparently it was the result of liquefaction of some small focus of tubercle left behind in the first operation. The little cavity was scraped and washed out, filled with iodoform emulsion, and closely stitched up. It healed up by first intention, and after some days the boy was sent to the country on a double Thomas's splint. He is here to-night, as well as could be desired.

About the same time I read a paper at Brighton at the request of the local Medical and Chirurgical Society on the technique which I had adopted in securing this result, which was only briefly alluded to in passing in my other communication. The Brighton paper was published soon after in the 'Brit. Med. Journ.' for January 19th, 1889, and as it was considered to be too closely allied in its text to my other communication, the latter was not included in the 'Transactions' of the Royal Medical and Chirurgical Society for the year.

The object of the present paper is to offer for the consideration of the Society eight fresh cases in which excision of the hip was performed since then on the same

principles, with hot-water flushing. I desire in the first place to describe somewhat in detail the state of the joints and surroundings in each of these cases. For, apparently, there has been some misconception as to the stage of the disease in which my first cases were operated on. In reference to this point I wish to state plainly that I have never yet excised a hip-joint in the earliest stages of tubercular destruction, and never before a large abscess or abscesses threatened to come through the skin, and the head of the femur or acetabulum, or both together, were extensively diseased.

I have advocated operation at a much earlier stage of disease than that at which it has been the custom to resort to excision hitherto. But that is a very different thing from advising operation at the very earliest stages ; and I have never for a moment lost sight of the numerous cases which must occur in every surgeon's practice in which rest and suitable general treatment have been followed by a subsidence of the tubercular process in the hip, even when at first it looked very unpromising.

The cases selected for the treatment to be described presently have always been those in which, in spite of all the care which could be bestowed upon them during months of treatment, the bone has been steadily invaded and abscesses have formed, advanced gradually towards the skin, and threatened to burst through it. These are surely rightly called advanced cases. In such, if excision is performed in the limited way advocated, we only anticipate what will inevitably take place soon, namely, the bursting of the abscess. But our opening into the latter is made under the most favorable conditions, and having made it, we simply carry out a recognised surgical principle in evacuating all diseased material within our reach, be it bone or synovial tissue. But the merit of the technique employed is that this evacuation is made so thoroughly that it is safe to close up the wound on the spot *without drainage,* and look for union everywhere in the healthy structures left behind, a sound cicatricial tissue taking

the place of the diseased area, and all open sinuses being prevented.

With the exception of one case uncompleted at that time (No. 2 in the appended table), the cases now to be described are the only ones in which I have excised the hip-joint since the paper alluded to was read at this Society on December 11th, 1888. One of them ought not properly to be included in this series, for in it the conditions were violated which I venture to think must exist if we are to obtain perfect results; for the skin in this case had been allowed to grow so thin and inflamed over the underlying abscess that its vitality was damaged, and union by first intention was not be expected. Moreover, the abscess had been aspirated beforehand probably with a non-sterilised needle, for the track of the latter suppurated independently. The joint, therefore, before the operation had true suppuration grafted on to the tubercular caseating process, and primary union in a suppurating wound was not to be looked for. But I have included this case in the series for completeness' sake and to avoid all cavil. The patient was at all events immensely benefited by the operation, although primary union of the wound did not take place. All the other six cases healed by first intention and have remained sound ever since, as you have seen. I had hoped to produce all seven cases to-night for your inspection, but the first boy, Case 3, I have been unable to trace from the time he left the convalescent home. This is perhaps due to the fact that his mother, a very worthless individual, had borrowed money from the sisters at the hospital and has kept away ever since.

In all these cases the technique of the operation has been the same. I should be sorry even to suggest that it is not capable of being improved upon. But after considerable experience of the older and newer operations, and of various methods of wound treatment, I know of none which seems calculated to give better results. Briefly described, it consists in opening the abscess round the joint by the anterior incision of Mr. R. W. Parker, then

introducing the flushing gouge which I have specially designed for the purpose, and thoroughly flushing and scraping the abscess with sterilised hot water. What remains of the head of the bone is then sawn off, extracted, and the cavity of the joint proper is flushed clean with the gouge which has again been introduced. Then the acetabulum is gouged until all diseased material has been removed, the *débris* being carried away by the stream of hot water rushing in and out vigorously during the whole procedure. Finally all the ramifications of the abscess cavity are similarly scraped, cut, and washed until the escaping water runs perfectly clear. The whole wound is then sponged dry and filled with iodoform and glycerine emulsion; then all the stitches are inserted, but before being tied any superabundant emulsion is pressed out. They are then knotted without any drain-tube being left in, pressure being kept up on every part of the wound until the last moment. Plenty of salicylic wool packed carefully round every part of the joint and thigh completes the operation, which will take about twenty to forty minutes. The child is at once placed upon a double Thomas's splint previously fitted, the limb being kept strongly abducted. How long it ought to remain upon this we do not yet know, but the time in my own cases has ranged from seven weeks to six or eight months. I am inclined to think that for a case of average severity four or five months will be the best time. They can leave the house on the splint in about a fortnight, and enjoy open air carriage exercise from this time on. When one compares these results with even the best secured by any of our older operative methods surely it must be admitted that a step in the right direction has been made.

The cases in the order of their occurrence are as follows:

Case 3.—James R—, æt. 7 years, a boy "who had never been strong," but had had no definite previous illness, was admitted into University College Hospital on Novem-

ber 1st, 1888. He was a thoroughly neglected, miserable-looking child, and came to us straight from Lambeth Infirmary. The mother was strong and healthy ; the father was not so, but had died of some injury to the bowel. About a year before admission the child was first observed to limp, and was treated for hip disease at one of the London hospitals for some weeks. From the onset he had severe pain in the joint, preventing sleep. The treatment lately at the infirmary had been weight extension, and a splint for a couple of months. When he came to us the right thigh was much flexed, abducted and rotated out. Over the joint in front and externally was much swelling ; the skin was reddened, the veins dilated, and there was deep fluctuation.

On November 2nd a large quantity of broken-down matter was aspirated from the abscess in the joint by one of my colleagues, and four ounces of iodoform emulsion was injected ; the patient was then placed upon a double Thomas's splint. After this the abscess soon filled again, and the skin became more thinned and discoloured.

On November 4th I excised the joint by the method and with the details described above. I had, however, but little hope of securing primary union in the wound on account of the damaged condition of the skin and underlying tissues, which were, as stated, inflamed, thinned, and discoloured. But I was anxious to see whether the flushing with hot water gave better ultimate results in the worst cases than our older methods of clearing out the diseased structures.

Both the head of the femur and acetabulum were found to be extensively diseased, the former being so deeply buried in the latter that for some time I thought there was a carious opening into the pelvis through which the remains of the neck had passed. But this was not the case, although the socket was very extensively diseased. What remained of the head was sawn off close to the trochanter, and the acetabulum was cut clean with the flushing gouge ; the latter was also used to cut and scrape all the diseased tissues from the affected area.

When the water ran clean the wound was closed as described without drainage.

The Registrar's description of the parts removed is as follows :—" These consist of the head of the bone. The central cancellous tissue is almost completely destroyed, so that the head consists only of a cup-shaped mass of soft bone. The articular surface is almost completely devoid of cartilage, being covered with soft, pinkish granulation tissue having around some remains of the cartilage. A large ragged strip of partly destroyed cartilage is quite separated, and lay loose in the acetabulum. No sequestrum, but a considerable quantity of spongy living bone was scraped from the acetabulum, together with a small amount of pale pulpy granulation tissue."

The day after operation there was considerable pain in the joint, and the temperature rose to 102·8° at 3 p.m. It then slowly fell to 101·6° at 11 p.m. Next day, when I dressed the wound and with a probe gave exit to ʒij or ʒiij of serum, it stood at 102·4° again. After this it fell again ; and though it rose somewhat for several days there was nothing in the boy's general condition to excite uneasiness. On the third day I dressed the wound once more, and found that much serum had soaked through. After a few days it was plain that the edges of the incision could not unite, and a boracic fomentation was applied. This was changed for a sal-alembroth gauze dressing a few days later.

All this time the patient was steadily improving, and on the sixteenth day he was taken out of bed and placed in a chair by the fireside. On the fifteenth day the aspirator puncture was found to be suppurating, suggesting that the joint had been infected by the needle in the first instance. A month after operation it is noted that the wound is " healing up well, though still discharging." At this time the child suffered from a splint sore, but gained strength nevertheless. On January 9th there was more discharge, and a probe went straight down for an inch and a quarter.

On February 15th the patient went to Eastbourne, having picked up considerably in flesh. There was occasionally a moderate amount of discharge in the middle of the scar.

When he returned from the convalescent home he had improved very much indeed in flesh and colour, but there was still a small sinus and a little discharge. We have been unable, as I stated, to trace this boy further in spite of several efforts.

This is the *only* case of the series *in which union by first intention did not take place.* The tubercular process, however, appeared to be extinguished by the operation, and from the state of the joint and sinus when last seen the part is probably healed by this time.

Case 4.—Isabella L—, æt. 9, was admitted into University College Hospital on September 11th, 1888. She was a fairly healthy-looking child, with no history of any illness except measles. The family history was also good. Nine or ten months before the patient fell upon her left hip and hurt it, and was consequently confined to bed for a few days. About two months before the *first* admission she began to limp, and the movements of the left leg were noticed to be restricted, with abduction and eversion; pain was also complained of at the same time in the hip, and later in the knee. On admission there was flexion, adduction, and internal rotation with marked lordosis. There was distinct fulness in front and to the outer side of the joint, but no pain on pressing the trochanter and no increased heat. The pelvis was tilted up on the left. There was no appreciable shortening. By extension with weight the deformity was corrected, and the patient was soon able to go home on a double Thomas's splint. The temperature was usually a little elevated, being, as a rule, above 99°. On November 19th, 1888, she was re-admitted, as the disease was manifestly extending. There was now more fulness over the joint in front, with tenderness on pressure. The trochanter was swollen, and on

pressing the bone towards the acetabulum it went in with a distinct jerk as if the head had been half dislocated, and grating was then felt plainly. On January 4th the fluctuation over the joint is noted as very distinct, and on this day I aspirated a quantity of curdy matter.

The abscess still increasing, I operated as described above on January 8th, 1889, six months after the onset of the disease. On opening the abscess about three ounces of its yellowish-brown contents spurted out. The head of the femur was sawn off close to the trochanter, and the extensively carious acetabulum was gouged clean with the flushing gouge carrying a stream of hot water. The abscess was now found to extend inwards under the rectus muscle and great vessels, and downwards in the thigh for about five inches. All its ramifications were thoroughly scraped and flushed at the same time, and much tubercular matter was evacuated. The wound was now closed without any drainage, and dressed after the usual toilet, a double Thomas's splint being applied with the limb abducted.

The parts removed are thus described in the Registrar's report :—"The cartilage is completely separated from the head of the femur except a few loose strips hanging round the margin. The surface of the head presents soft vascular rarefied bone, covered in parts with pale, soft, pulpy granulation tissue. The saw section of the neck appears normal, but is very vascular."

Little more need be said ; the wound healed by first intention without any complications. The highest temperature was 101·2° on the day after operation. The first dressing was on the ninth day, the stitches were all removed on the fifteenth day, and the patient left for the convalescent home two days later. When she returned she was in excellent health, and there was no tenderness or pain about the joint. In May she was shown by Mr. Raymond Johnson at the Clinical Society for me ; there was then considerable movement in the joint, and no deformity except slight shortening. She was kept on the splint for about eight months and then allowed to walk,

which she now does well. There is about an inch and a quarter shortening, and a slight tendency to rotate the toe outwards, and very slight flexion and adduction hardly noticeable.

In this case the operation was watched with interest by several surgeons of large experience, including Mr. Pick, Mr. Croft, Mr. Macnamara, and Mr. R. W. Parker, and Dr. Whittle, of Brighton, who were particularly struck by the extent of the disease and the awkward position of the abscess under the great vessels of the thigh.

The next two cases I was invited to operate on by Dr. G. Whittle at the Alexandra Hospital for Children at Brighton, to demonstrate this method of procedure to a number of his medical friends.

The first of them (George L—, Case 5) I thought was in a most unfavorable condition for testing the method. The patient was a very unhealthy boy with a bad family history. For six months he had been suffering, and for three of these there had been an abscess in and around the hip-joint. When I first saw him I demurred about subjecting the method to so severe a test, especially as I was particularly anxious to demonstrate the benefits of primary union in these first cases. The thigh was greatly enlarged, and the skin was red and congested ; the state of things was more that of an acute abscess than what we usually see. I explained that it was highly probable that the wound would not heal or would soon break down again. However, the operation was performed just as in the last case. But there was much more difficulty in clearing out the tubercular matter, which had more widely infiltrated the tissues round the joint than in any previous case. It was particularly difficult from the fact that the tubercular infiltration was still in great part semi-solid, not having everywhere undergone liquefaction. The head of the femur and acetabulum, which were extensively diseased, and all the infected area, were gouged and flushed. The rest of the treatment was as in the other cases. The parts removed are preserved.

When I next saw the boy on the fourteenth day, up to which time the dressings had not been touched, the whole wound was soundly healed without a drop of pus, and has remained so ever since.

Case 6. Winifred S—, æt. 5, did not look in so unfavourable a condition as her fellow patient. But as it turned out besides a large anterior abscess the acetabulum was destroyed and perforated so that I could put my index finger into an abscess within the pelvis, and then discovered a small loose sequestrum the size of the tip of the little finger. The head of the femur was half separated already from the neck, besides having nearly all its cartilage loose on its carious articular surface. This operation was done like the last. The anterior abscess was found to extend for several inches down the thigh ; when it had been treated the joint was attacked. Then the neck of the femur was sawn through and the head was removed. The acetabulum was next widely gouged, especially the hole at its fundus leading into the pelvic abscess. Through this aperture the gouge was introduced, and the cavity was scraped thoroughly and flushed with the hot water at the same time. The sequestrum (shown) came away with the latter. Other details as described. The parts removed are preserved.

I next saw the patient on the fourteenth day after operation and removed the first dressing. The wound was soundly healed without a trace of discharge, and has remained so up to the present.

Case 7. George J—, æt. 11 years, was admitted into University College Hospital for the present affection on April 10th, 1889, having previously attended the Out-patient Department for some months. His family history was not good, and he himself has nearly always had a cough, and at one time is said to have suffered from "consumption of the bowels."

Six months before admission the patient began to limp,

and complain of stiffness in the left hip on walking. He became at the same time very thin and weak. On March 11th I had him placed upon a double Thomas's splint. On April 3rd a swelling was noticed on the front of the thigh, below the anterior inferior spine of the ilium. When admitted he complained of pain in the upper part of the thigh. The only trace of deformity was a little adduction with slight elevation of the pelvis on the affected side. There was distinct fulness in front and to the outer side of the joint, and the hollow behind the trochanter was slightly filled up. No fluctuation could be obtained in the swelling, nor was there pain on striking the heel. But when the trochanter was pressed inwards he complained. There was considerable limitation of flexion, but no alteration in length of limb. On April 28th he was sent to the convalescent home on a double Thomas's splint. Up to July 18th there was no increase in the swelling, but when seen on September 2nd, enlargement of the abscess, fluctuation and redness of the skin, with dilatation of its veins were discovered by Mr. Johnson, who was seeing my out-patients. The limb was now fixed in slight abduction, and there was half an inch shortening. As the abscess threatened to burst, he was re-admitted under my care, and on seeing him it was determined to anticipate this by operation. On September 3rd excision was performed as in the other cases above described. A large abscess was opened first, which extended into the joint, and down the thigh from under the tensor vaginæ femoris on the outside, across to its inner side superficial to the rectus muscle. In the joint there was a large amount of tubercular matter. The head of the bone was sawn off, and the acetabulum, which was very extensively diseased, and hollowed by large pits, was gouged and flushed. The remaining details were as in the other operations.

The highest temperature after this was 99·4°, and there was no pain. The stitches were removed on the thirteenth day; complete union by first intention was found

everywhere. The wound has since remained quite sound, and the boy has grown fat.

The head of the bone was so diseased as to crumble away during removal; no detailed description of it, therefore, can be given.

Case 8. Emily S—, æt. 9, a fairly healthy-looking child, with a good family history but very bad hygienic surroundings, was admitted into University College Hospital on February 19th, 1890. Eleven months before she had been an in-patient with symptoms of hip-disease on the right side, *i. e.* limping, and screaming at night. In the preceding October she was again re-admitted on account of swelling over the joint, which has been steadily increasing in spite of continuous treatment on a double Thomas's splint.

On admission on February 19th, 1890, there was considerable general swelling about the right hip but no œdema, redness, or fluctuation. There was slight flexion and distinct adduction but no rotation, There was pain on passive movement. Temp. 99·4° to 100·4°.

On March 11th it is noted that the "general tenderness and to some extent the general swelling has subsided," but "below the outer half of Poupart's ligament deep fluctuation can be distinctly felt and there is slight œdema with some dilatation of the superficial veins, but the deepest pressure gives rise to no pain."

During May she began to cry out frequently during sleep, and at the end of the month there was half an inch shortening (there was no shortening on admission), the abscess was also more fully developed.

On May 23rd I excised the joint as described, evacuating first about three ounces of curdy material, and then removing the head of the femur and the diseased tissue of the acetabulum which was pitted all over with cavities containing granulation tissue. After the usual flushing the cavity was filled with iodoform emulsion and completely closed without a drain-tube.

The head of the femur was found to have its "cartilage destroyed except a few spots around its periphery." The "soft cancellous bone was exposed and covered with granulation tissue."

The first dressing was on the twelfth day, when perfect primary union was found and the stitches were removed. A few days after this she had measles, but without any ill effect on the joint, which has remained soundly healed until now.

The temperature during the first twelve days after operation only once touched 100·6° and then became normal.

Case 9.—Ellen T—. Left hip diseased about twelve months before operation. Double Thomas's splint worn for about four months. Abscess began to form in front of joint some months before operation, and steadily advanced.

On July 4th, 1890, excision was performed as above. The head was found extensively diseased, and the acetabulum much eroded. In the latter lay a small loose sequestrum the size of the tip of the finger. The head was removed, and the whole cavity gouged and flushed clean. Union was practically *per primam*, and the child left hospital on August 2nd. She is now quite well and fat, but still wears a double Thomas's splint. The highest temperature recorded in this case was 100°.

It may be suggested that these results are exceptional, and that we must not expect them to be the rule. This may be true if cases with open sinuses are operated on by this method; then, of course, we shall not get lasting union by first intention. And in instances in which the incision has to be made through thin blueish skin we shall probably fail to obtain such results. But if we take patients in whom there are abscesses, however large (and in some of the cases before you they were very extensive) provided that the skin and subcutaneous fat be fairly

healthy, I do not see why we should not obtain as good healing in every instance. If it is possible to do so in a large proportion of cases now, it ought to be so in all as we go on improving the details of our technique. At all events it behoves us to try. And I hope to be able hereafter to justify the forecast made in my former communication to this Society, that before long we should have whole series of cases of excision of advanced hip disease where union by first intention had been obtained; and without any mortality. As a step in this direction I venture to bring forward this short series of cases to-night, and in doing so I promise that the patients shall be kept as far as possible under observation, and as long as possible, and that I shall ask permission to produce them here, whether well or ill, on some future occasion.

I do not expect to present them with the affected limb as long as its fellow; this would be impossible. Many cases of hip disease which have recovered without any abscess and without any operation show a stunted, shortened member. And if in addition to disease which has softened the head of the femur and acetabulum an operation has been performed we must of course have shortening. But I would like to point out that if the operation is done before all the capsular structures are destroyed by the disease we may expect a minimum of shortening. For this procedure as described does not necessarily damage the capsule except to the smallest extent. In it the anterior fibres are divided mainly outside the Y ligament where they are thin and unimportant. The rest of the capsular structures, if the disease have spared them, are left intact and capable of giving support to the femur. Nor are the muscles, nerves, or, more important still, the vessels injured in any way so that the nutrition of all the parts to be repaired is not impaired in the least.

In conclusion I may be permitted to reiterate that this procedure is not advocated to supersede the treatment by rest, general and local, together with the other usual remedies, for which I am the strongest advocate in all

cases so long as it can be carried out with benefit. It is suggested rather as a means of treating tubercular hips where these other measures have failed. And it is to be hoped it may supersede those older methods of excision which were not only immediately dangerous to life, but which, as we all know, gave most unsatisfactory ultimate results as to progression and symmetry. That the procedure is not yet by any means perfect is obvious; but having had many heart-burnings in the past regarding the former treatment of this large and distressing group of diseases, I offer this procedure as one which has given me for my own part better results than any which I have ever seen put in practice. It is, at all events, simple and safe, and gets rid of the use of dangerous germicides and of shock. I venture to think, therefore, that it is worthy of further trial.

The days of bad immediate results after excision of the hip are, it is to be hoped, past. Aseptic surgery has practically got rid of the wound infections which used so frequently to follow the operation speedily. And I hope, so far as my own practice is concerned, to have got rid by the method now advocated, of the chronic sinuses which were so frequently a source of danger.

I will only add in conclusion that for my own part all the dreary forebodings which used to occur to one when confronted by a case of destructive tubercular hip disease have ceased. Such a case can now be approached with tolerable certainty that the local disease can be thoroughly eradicated, and that the wound will be soundly and permanently healed in three weeks. We have still an immense amount to learn, but this is at all events something.

But I cannot close this paper without expressing the hope that we are not far from the time when the necessity for any operative interference with tubercular joints will be obviated by a more general recognition of the disease in its earliest stages, and a more intelligent and thorough treatment by rest, &c.

But so long as we have to operate on neglected cases, let us aim at doing so with the minimum of risk, suffering, and loss of time to the patient, and the maximum of benefit to the limb and general health.

Whether the method now offered for your consideration will assist us in this aim is a question which may be safely left for the friendly and impartial verdict of this Society.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 4.)

No.	Sex, age, date of operation, name.	Duration of subjective symptoms.	Duration of objective symptoms before operation.	State of parts found at operation.	Immediate result of operation.	Ultimate result of operation.
1	M., 5 years, July, 1888, right hip, Lawrence Taylor	12 months; splint worn 10 months before operation	Some months, during which large abscess formed in front of joint. No appreciable shortening	Anterior incision. Head of femur extensively eroded, and acetabulum also. Head removed; acetabulum gouged and flushed. No drain tube	Healed by first intention. Sutures removed on 13th day, and patient sent home on D. Thomas's splint	Splint removed 8 weeks after operation, and patient began to walk. 12 months later, patient running about without pain. Considerable mobility of joint; slight tendency to flexion. Wound soundly healed, *vide* p. 40. Patient shown at meeting quite well, and remains so.
2	M., 6 years, July, 1888, right hip, Alfred Cannon,	12 months; D. Thomas splint worn 12 months	Duration not known. Large abscess in front of joint. No appreciable shortening	Anterior incision. Head of femur extensively eroded; acetabulum bare. A tubercular focus on sawn surface of femur gouged and flushed. Drain tube for 24 hours	Healed completely on 10th day, when stitches were removed, and patient was removed to Children's Hospital. The last dressing had been allowed to become soaked with urine and to become putrid. In this way the stitches were inoculated with septic matter, and ultimately suppurated in Children's Hospital	7 weeks later readmitted with sinus at seat of drain tube. 17 months later this was dilated, and the acetabulum was found perforated into an abscess, which drained into the rectum. The other hip at this time also contained a large abscess. Not long after the child died of general tuberculosis and marasmus.
3	M., 7 years, 14th Nov., 1888, right hip, James Ready	12 months; splint worn only for 12 days before operation	12 months, during which large abscess formed in front of joint. Skin very much thinned and discoloured	Anterior incision. Head of femur eroded extensively, leaving very little to be removed; acetabulum deepened by erosion, and osteophytes around margin gouged and flushed. No drain tube	Healed, but wound slowly gave way again, the skin being so thin; an opening also found at old aspirator puncture	Boy got fat and strong, but still had small sinus, with a trace of discharge, when he left for the country. Cannot be traced since.
4	F., 9 years, 8th Jan.,	6 months; D. Thomas splint worn	4 months, during which abscess formed over front of	Anterior incision. Head of femur and acetabulum extensively diseased. Head	Healed per primam. Sutures removed on 14th day. Left hospital on	Wound remained soundly healed. Wore splint for over 6 months, then began

	left hip, Isabella Lowen	before operation	joint. The head of femur had slipped partially out of socket, and could be pushed in with a click	removed and acetabulum gouged clean and flushed. The abscess extended down the thigh and under the femoral vessels to the inside. It was thoroughly scraped and flushed. No drain tube	16th day wearing D. Thomas's splint	to walk about. 10 months after operation was in excellent health, and the hip was soundly healed. The limb was shortened about ¾ in., and the foot slightly everted; the limb was perfectly straight, and there was considerable mobility of hip. Cannot be traced.
5	M., 5 years, June 8th, 1889, left hip, Geo. Leaney	6 months	Over 3 months. Large abscess on upper and outer side of thigh. 1 in. shortening	Anterior incision. Not much pus in the swelling, chiefly pulpy matter. Most of bone disease in neck of femur; acetabulum eroded. Head of femur sawn off, the rest gouged and flushed: no drain tube. D. Thomas's splint	Sutures removed 14th day; wound healed by first intention. Left hospital well in 3 months, wearing D. Thomas	24 months after operation, joint looks quiescent. Wound quite sound when patient shown at meeting.[1]
6	F., 5 years, June 8th, 1889, right hip, Winifred Standen	9 months	6 months. Fulness about joint. No shortening. Deep seated fluctuation	Anterior incision. Large deep abscess opened. Head and neck of femur diseased, also acetabulum, and there was a perforation here about the size of forefinger into the pelvis. Head of femur sawn off, rest gouged and flushed: no drain tube. D. Thomas's splint	Suture removed 7th day; wound healed by first intention. Left hospital well in 4 months, wearing D. Thomas	Wore D. Thomas's splint, 6 mo. after operation. Looked remarkably well, and had grown very fat. Hip quite sound. Patient shown at meeting. In Dec., 1890, child received an injury of the hip, and end of Jan., 1891, a large abscess was found over outer aspect of limb about to burst; child walked about still notwithstanding. Abscess opened and flushed by Dr. Whittle. He writes, May 31st, 1891, that all is soundly healed and child is quite well.

[1] End of March, 1891, a mass of caseous material discovered under the skin over outer aspect of joint by Dr. Whittle, who excised it and obtained primary union of resulting wound. Writing May 31st, 1891, he says all is sound and the boy is in excellent position.

No.	Sex, age, date of operation, name.	Duration of subjective symptoms.	Duration of objective symptoms before operation.	State of parts found at operation.	Immediate result of operation.	Ultimate result of operation.
7	M., 11 years, Sept. 3rd, 1889, left hip, George Jeans	11 months; D. Thomas splint worn 6 months before operation	5 months before operation an abscess began to form in front of joint. ½ in. shortening. Cutaneous veins dilated, and skin reddened over the abscess	Anterior incision. A large abscess cavity opened over the joint, extending under tensor vag. femoris and over origin of rectus. Head of femur much eroded, sawn off, and acetabulum extensively diseased; gouged and flushed: no drain tube	Healed per primam, and remained firmly healed. Stitches removed on 12th day. Left hospital on a D. Thomas's splint on 17th day quite well; limb perfectly straight	Boy rapidly improved and became fat. About a month or so after operation there was apparently some simple thrombosis of one of the large veins of the thigh, but this soon passed off, and gave rise to no trouble beyond swelling. Patient shown at meeting. Seen several months later quite well.
8	F., 9 years, May 23rd, 1890, left hip, Emily Slattery	14 months; splint worn 8 months before operation	8 months before operation an abscess began to form round the joint	Anterior incision. Abscess flushed with hot water; diseased head removed; diseased acetabulum gouged and flushed: no drain tube. Iodoform emulsion. Complete closure of wound	Good, no complications. 1st dressing on 12th day. Perfect union by first intention	Patient on D. Thomas's splint for 6 months. Patient shown at meeting well, with hip sound. Cannot be traced.
9	F., 4 years, July 4th, 1890, left hip, Ellen Tree	12 months; splint worn about 4 months	Some months before operation abscess began to form over the front of the joint	Anterior incision. Abscess opened and flushed. Diseased head removed; sequestrum loose, removed; acetabulum diseased, gouged out and flushed: no drain tube. D. Thomas's splint	Stitches removed 11th day; union by first intention. Left U. C. H. August 2nd; wound soundly healed	Wore Thomas's splint for 6 months. Patient shown at meeting. Seen April, 1891, fat and strong, beginning to walk, leg quite straight. Splint only worn at night.

Last column corrected down to date, June, 1891.

SOOT IN CELLS OF CHIMNEY-SWEEP'S CANCER.

BY

W. G. SPENCER, M.S.

Received June 7th—Read November 11th, 1890.

Since Pott[1] first described the disease it has always excited interest out of proportion to its relative frequency, and surgeons, especially in London, have recorded many cases. Two papers by Earle,[2] and Travers[3] respectively, are to be found in the Transactions of the Royal Medical and Chirurgical Society. The interest lies in the fact that from a known cause, viz. from soot, a new growth may be produced in some persons, which has the anatomical structure of epithelioma, and which may extend to the neighbouring lymphatic glands, but no further. Moreover this purely local disease is connected with ordinary epithelioma of the malignant type by other cancers[4] of somewhat similar causation, viz. that occurring among workers

[1] Pott's Works, ed. by Earle, 1808, vol. iii, p. 178.

[2] Earle, 'Med.-Chir. Trans.,' 1823, vol. xii, p. 296.

[3] Travers, ibid., 1828, vol. xvii, p. 344.

[4] von Volkmann, "Ueber Theer u. Russ Krebs.," 'Verhandlung der Deutchen Gesellschaft für Chirurgie,' 1874, iii Congress, Berlin, i, s. 3.

in tar,[1] and paraffin,[2] and that from chewing or smoking[3] tobacco.[4]

I shall, in this paper, confine my remarks to the points illustrated by the microscopic specimens, and this I do the more readily because Mr. Butlin is following in the footsteps of his predecessors at St. Bartholomew's Hospital, and has collected all the general information on the subject, both from published and unpublished sources. From him, therefore, we may expect valuable additions to our knowledge on the subject.

I have not been able to find that any previous observer has described the presence of soot particles in chimney-sweep's cancer.

The case from which the specimens were taken is a master-sweep, aged sixty-three, who formerly lived at Kingston, and is now in Croydon. In March, 1885, an ulcer, which is said to have been epitheliomatous in structure, was removed from over the left zygoma by Mr. Macnamara in Westminster Hospital. Although he comes from a family of sweeps, he is not aware of any members having suffered in a similar way. In August, 1887, he first came under me. The skin was dry and harsh, there was some eczema over the body, and in some parts dusky patches, but in others the skin was free from any pigmentation. There was a black ring in the skin round the neck, at the level of his collar, in which ring to the left of the middle line behind was an indurated ulcer about an inch and a half in diameter. At the level of his waistband was a similar ring, and in it, over the erector spinæ of one side, was another indurated ulcer two inches long by one inch broad. There was also a small patch of superficial ulceration on his left cheek, at some distance from the scar of the first operation.

[1] Ball, "Tar Epithelioma," 'Lancet,' 1885, i, 1037.

[2] Ogston, "Local Effects of Crude Paraffin," in 'Edinburgh Med. Journ., 1871, Dec., p. 544.

[3] Esmarch and Langenbeck, von Volkmann, Discussion.

[4] Tillmann's, "Ueber Theer Russ v. Tabakskrebs," 'Deutsche Zeitschrift für Chirurgie,' 1880, xiii, 519.

The two large ulcers were excised and immediately placed in alcohol, and the cheek was scraped.

In April, 1890, he returned with superficial ulceration again on his left cheek, about two inches square, which had the appearance of lupus. Scraping was once more done. With this exception he has remained quite well and there has been no affection of lymphatic glands.

Both the ulcers and the product of scraping were found to present the same appearance microscopically, and about one hundred preparations have been examined; most of the points mentioned below are to be seen in each of the sections. Various stains were used, including logwood, but it is difficult to observe the black cells in a specimen stained with the last-named dye, until the eye has become familiar with lighter stained specimens. Perhaps the use of logwood may have caused the appearance to have been overlooked, as certainly happened to me when I stained the first sections in this way.

Microscopic Sections.

Specimen I. (Fig. 1.)—A vertical section through the ulcer, to show the general structure; stained in safranin. The growth is an epithelioma, portions being of the squamous-celled variety, with here and there an incomplete nest-cell, whilst other parts have rather the usual appearance of rodent ulcer, and consist of smaller oval cells. At the margin the disease was in course of extension by means of columns of cells growing downwards from the deeper layers of the epidermis, and more advanced columns have become flask-shaped and surrounded by leucocytes which have collected in the neighbouring tissues.

Specimen II.—An unstained section, which was immersed in 33 per cent. nitric acid for twenty-four hours. Black masses appear under a low power, scattered throughout the section, and collected in heaps. Under a high power the black material is observed to fill both large

cells and small cells. The outlines of the cells are clearly mapped out by the black material which they contain.

Specimen III. (Fig. 2.)—The section was stained in cochineal. Under a high power the growth appears to be composed of oval cells, amongst which spaces occur, and around which spaces the cells have a tendency towards a columnar shape. Between the oval cells and in the spaces are scattered large cells with single nuclei and a large amount of protoplasm, which have become filled with black granules, so that in the case of many of them little of the protoplasm of the cell is to be seen. A few small cells are also present, being likewise stained black by the material which fills them.

Specimen IV. (Fig. 3.)—The section was stained with picro-carmine, and is seen under a high power. At the margin of the growth, in the corneous layers of the epidermis there are soot particles, and among the cells of the rete Malpighii are some darkly stained by the black material within them. From the lower layer of these cells two columns have begun to grow down, and some of the cells in these columns are stained black by granules similar in appearance to the soot in the corneous layers.

Specimen V. (Fig. 4.)—The section was stained in safranin. Under a high power the still healthy epidermis, immediately beyond the margin of the growth, has the lower cells of the rete Malpighii, between the papillæ of the corium, filled with black granules.

Specimen VI. (Fig. 5.)—The section was stained with safranin and picric acid. A column of cells, seen under a high power, has grown from the epidermis and divided into groups of cells, some of which are filled with soot particles. Several of the cells appear to have a large amount of protoplasm, and to be in course of becoming the large cells seen in Specimen III.

Specimen VII. (Fig. 6.)—The section was faintly stained in primrose yellow. Under a high power a loosely formed nest-cell appears, having soot particles between its

concentric layers. Beside it is a portion of the growth, amongst the cells of which are some coloured black.

Specimen VIII. (Fig. 7.)—The section was stained with pale primrose yellow. The deep portion of the growth is seen, under a high power to be composed of cells which are mostly oval, but which have a more columnar appearance at the margin. This is the part of the growth which was extending deeper into the structures beneath. Amongst the cells of the growth are numerous smaller cells coloured black, especially occurring amongst the columnar cells of the margin.

Specimen IX. (Fig. 8.)—The specimen was stained in pale safranin. Immediately outside the new growth, among the leucocytes and newly-formed inflammatory tissue are numerous cells, some large, some small, coloured black by the soot which they contain.

Specimen X.—Part of the growth as seen with the naked eye. The cut surface shows dusky patches.

The foregoing specimens show soot in the corneous layers of the epidermis, in the cells of the rete Malpighii, in the growing ends of the epithelial columns which begin the growth, in the cells of a column which has become flask-shaped and is tending to separate into groups of cells, and in the nest-cells which arise in the epithelial columns. Cells full of the same material, some large and some small, are found in numbers in the centre of the growth, at the deep growing margin, and in the tissues outside.

The soot can be traced into and around the growth by means of its black appearance, and the appearance is unaltered by treating the sections for twenty-four hours with 33 per cent. nitric acid, or with nitro-hydrochloric acid. Except for the soot particles in the corneous layers, all the rest is within cells. In some places, no doubt, a cell has been broken by the microtome, and the black granules are outside, but it can be easily seen that this extra-cellular position has been produced by the destruction of a cell.

Also here and there a black particle has fallen from the atmosphere in which the specimens were mounted. With this exception, and the contained soot, the sections have never been in contact with any other carbonaceous material.

The large cells which contain soot resemble the cells of the epithelial columns from which they appear to be derived, whilst the small cells are like the oval cells which compose the centre of the growth. The specimens, therefore, indicate that the cells containing soot are epithelial in origin.

Carbon and carmine have been shown to be taken up by cells and carried to the lymphatic glands, whether this be from a spot in the skin which has been tatooed, or from the lungs, *e. g.* of colliers, to the bronchial glands. Some[1] have stated that the cells which take up carbon are epiblastic in origin, others that they are mesoblastic.

In the present case the epiblastic origin of the black cells seems fairly certain, for the soot is manifestly in the cells of the epithelium to begin with, and there is no sign of any transference of the soot to leucocytes—*e. g.* by the epithelial cells breaking down.

These specimens, I think, offer simple explanations of some points in the clinical characters of chimney-sweep's cancer which have hitherto been obscure.

(1) These large cells having taken up soot which has entered the skin are to be found outside the growth and may travel to the neighbouring lymphatic glands, just in the same way as is the case with cells filled with innocuous carbon, and there set up in the lymphatic glands chimney-sweep's cancer. We should then have an explanation afforded of the way in which soot cancer may develop primarily in the lymphatic glands. Mr. Paget[2] removed, from a sweep aged forty-eight, the in-

[1] Virchow, "Ueber Lungenschwarz," 'Virchow's Archiv,' Bd. xxxv, p. 348; Tchistovitch, "Des Phénomènes de Phagocytose dans les Poumons," 'Annales de Pasteur,' 1889, Juli, p. 334 (with Bibliography).

[2] Paget, 'Med. Times and Gazette,' 1852, vol. ii, 414.

guinal glands on both sides. There was no other lesion except a dry and dusky skin. Mr. Lawson[1] has described two such cases. In one, a sweep, aged fifty-six, the left femoral glands formed a hard swelling which afterwards ulcerated continuously until the superficial femoral artery was opened. The skin was dusky from ingrained soot, but there was no scrotal lesion. In the second case the inguinal glands were attacked and ulceration took place into the external iliac. In my patient the glands have as yet remained unaffected.

(2) The presence of soot in the lower cells of the epidermis, and in the tissues outside the growth explains the dusky patches in the skin of sweeps to which Pott,[2] Astley Cooper,[3] and others since then have called attention—dusky patches which no amount of soap and water will remove. In other parts the skin may be normally white as in my patient. Earle[4] had seen many cases of ulceration on the cheek, and Travers[5] likened the appearance to lupus. The growth at the line of the collar and waistband in my patient may be compared with Lawrence's[6] case on the ear from contact with a soot-bag.

(3) The presence of the soot in the skin and tissues suggests that it may remain latent there until "predisposition" favours the starting of cancer. If that be so, we have an explanation of the fact which has until now proved puzzling, viz. the development of chimney-sweep's cancer years after the patient has ceased to come in contact with soot. In one of Earle's[4] cases a man aged twenty-eight had a bubo which suppurated; then a fungating ulcer formed, which took on all the characteristics of the disease. He had been a sweep when a boy, until thirteen years old, and during that time had soot warts

[1] Lawson, 'Lancet,' 1878, vol. ii, 577.

[2] Pott's Works, ed. by Earle, 1808, vol. iii, 178.

[3] Astley Cooper in 'Observations on the Testis,' 1830, p. 226, "Of the Chimney-Sweep's Cancer."

[4] Earle, 'Med.-Chir. Trans.,' 1823, vol. xii, 296.

[5] Travers, ibid., 1828, vol. xvii, 344.

[6] Lawrence, quoted by Paget, 'Lancet,' 1850, vol. ii, 265.

on the scrotum and groin. But for the last fifteen years he had not been in contact with soot, nor had he suffered from any scrotal cancer. Curling[1] records the case of a sailor, between forty and fifty, who had been a sweep in boyhood, but for twenty-two years had been at sea, out of all contact with soot, yet a growth occurred on the scrotum having all the genuine characteristics of a chimney-sweep's cancer.

In a case under Stanley[2] the man was a sweep until twenty-one, when the occurrence of a large ragged soot wart on the scrotum so frightened him that he left the work and became a hawker, in which trade he was not in the least exposed to soot. Yet the growth returned six years after and was removed by Lawrence. After another five years it returned again, but grew so slowly that it was not removed by Lloyd until he was thirty-nine. Finally the glands became affected and Stanley removed them with the overlying skin.

The presence of the soot in the skin further explains why recurrence may take place, not in the scar, but at some distance from it. This has often happened on the scrotum and elsewhere. In a sweep, aged sixty-six, under Curling,[1] a scrotal cancer had been removed thirty-six years before, later on a finger was amputated for the same reason, and later still a soot-wart appeared on a finger of the other hand.

(4) No doubt another factor besides the soot is required for the above explanations, viz. predisposition. But predisposition in the case of soot-cancer is not entirely an hypothesis, for there is evidence to support it at least on two points, viz. as regards age and as regards inheritance.

Earle[3] said that although eczema and soot-warts were common enough among the boys, yet cancer did not

[1] Curling, 'Diseases of the Testis,' 1856, 2nd edit., "Chimney-Sweep's Cancer."

[2] Stanley, 'Med. Times and Gazette,' 1852, vol. ii, 414.

[3] Earle, 'Med.-Chir. Trans.,' 1823, vol. xii, 296.

develop until after twenty and generally not until after thirty. Only this age limit appears to have been diminished when an inherited predisposition was also present. In one case only is cancer recorded in a boy, viz. by Earle, sen., in a boy of eight years.

Inherited predisposition was well marked. Earle saw instances of three generations—father, son, and grandson attacked. Hawkins[1] had a father and son under him at the same time for the disease. Cusack[2] removed a soot-wart from the hand of a woman who carried on a chimney-sweep's business. Twelve years before he had removed from the ear of her son a soot-wart, and another son had died of cancer.

It is obvious that in the great majority of sweeps this predisposition is absent and they remain immune.

(5) There is one point of general interest which the specimens show. Cells filled with the soot are seen among the leucocytes outside the margin of the growth. These black cells are epithelial in origin and therefore form the advancing portion of the disease. It has been for long an axiom in clinical surgery that a wide margin must be removed beyond the apparent margin of the growth, as seen either by the naked eye, or by the microscope. But it is impossible, as a rule, to identify the cells of the growth when amongst the leucocytes outside the well-defined margin. Here the black colour enables us to do so and thus to give an anatomical support to the surgical axiom of removal wide of the disease.

[1] Hawkins, 'Lond. Med. Gazette,' 1838, vol. xxi, 842.

[2] Cusack, 'Dublin Journ. of Med. Science,' 1842, vol. xxi, 137.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 12.)

DESCRIPTION OF PLATE I.

Soot in Cells of Chimney-Sweep's Cancer (W. G. Spencer, M.S.).

Fig. 1.—General structure of the growth. 1. Epithelial cells. 2. Epithelial columns. 3. Portion of growth composed of oval cells.

Fig. 2.—Large and small cells in the centre of the growth filled with soot.

Fig. 3.—Soot in the corneous layers of the epidermis, in the cells of the Malpighian layer, and in those of the epithelial columns.

Fig. 4.—Epidermis beyond the margin of the growth. Granules of soot are contained in the cells of the Malpighian layer.

Fig. 5.—An epithelial column which has divided into groups of cells, some of which contain soot.

Fig. 6.—Epithelial nest-cell with soot amongst its concentric laminæ.

Fig. 7.—The deep growing margin, showing many cells coloured black by the soot particles which they contain.

Fig. 8.—Soot contained in cells outside the apparent margin of the growth. 1. The growing margin. 2. Inflammatory small round cells.

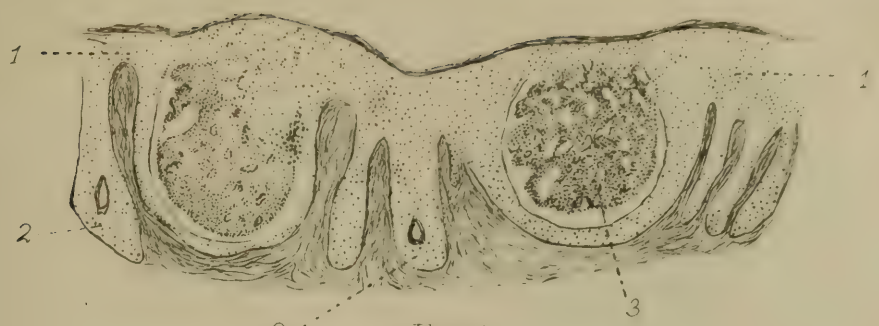

Fig 1

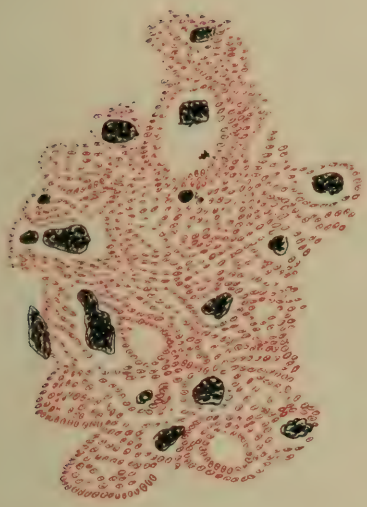

Fig. 2

Fig. 3.

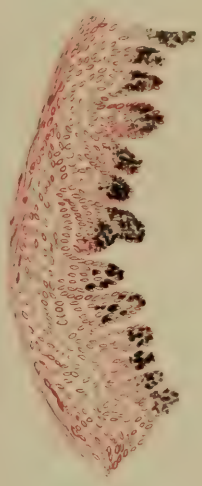

Fig. 4

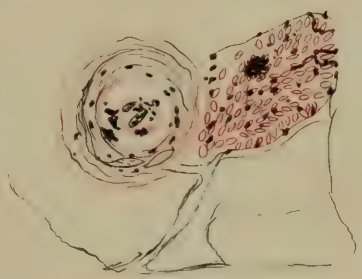

Fig. 6.

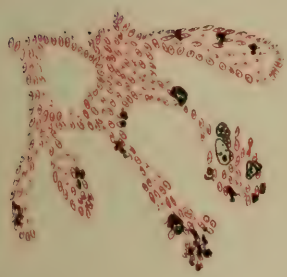

Fig. 5.

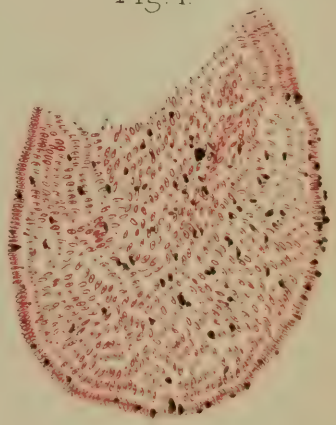

Fig 7

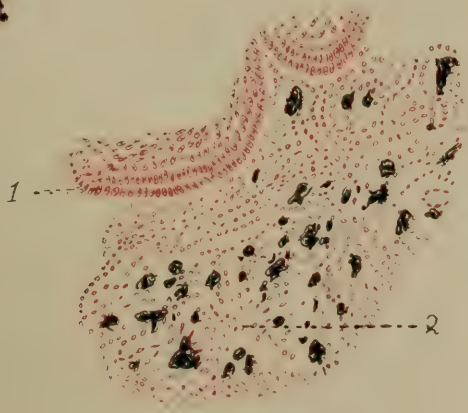

Fig. 8.

Danielsson & Co. lith

SUPPURATIVE AFFECTIONS OF THE TESTICLE AND EPIDIDYMIS.

BY

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Received June 18th—Read November 11th, 1890.

Abscess in the testis or epididymis is of infrequent occurrence. The majority of our text-books do not treat of the subject as worthy of distinct and separate consideration. So distinguished and experienced an authority as Curling, only mentions the possibility of acute inflammatory affections of these parts terminating in suppuration, and the authors of well-known surgical articles in standard works allude to the subject in the same manner, noticing the occasional termination of both acute orchitis and epididymitis in suppuration.[1]

An investigation of the matter has, on the whole, only confirmed the view taken by various authorities of the comparative infrequency of testicular abscesses. There are, however, recorded instances of suppurative affections of these organs scattered throughout medical literature. Some cases also have occurred in the practice of surgeons. It is hoped that a collective consideration of them, and of

[1] Holmes' 'System of Surgery,' 3rd edit., vol. iii, pp. 515, 527.

the subject generally, in a brief paper, may not be devoid of interest and utility.

Many French authors refer to cases of suppuration of the testicle. Terillon and Mounod, in their recently published work, refer to instances occurring after the specific fevers, and from tuberculous disease. They quote an observation of Sédillot, that an acute inflammation of the sac of a retained testis may terminate in suppuration, and assert that puncture with a trocar may produce a like result. Gosselin[1] devotes the greater part of a lecture to the consideration of cases of orchitis, which terminated in suppuration and destruction of the testis. I can find no records of such cases in the Transactions of the London societies. There is no marked recognition of them in the surgical reports of the larger hospitals, though instances are noted by the registrars in the tables of surgical cases. Royes Bell, writing in Ashurst's 'Encyclopædia,' states, that in fourteen cases of acute orchitis following urethral operations, five suffered from acute abscess which required evacuation by incision. The well-known softening and disintegration associated with syphilitic deposit in the testis is recognised, and referred to by most authors. Some specimens described as "Abscess of the Testis" in the Museum of St. Thomas's Hospital are apparently of this nature.[2]

It would seem that so far as causation is concerned abscess of the testicle and epididymis may be considered as follows: (1) A considerable proportion of the cases will be found due to the softening and disintegration of tuberculous or syphilitic material within the organ. (2) Suppuration of the testis occurs as a rare termination of gonorrhœal inflammation. (3) Orchitis, complicating operations upon the bladder and urethra, may terminate in suppuration. (4) A certain proportion of cases are noted as a sequel to the specific fevers, especially typhoid, and as the result of pyæmia. (5) Abscess may follow injuries to the testicle, as blows, crushes, or puncture. (6) Lastly, certain

[1] 'Clinique Chirurgicale,' tom. iii, p. 389.

[2] St. Thomas's Hosp. Museum, E. E., 13, 15.

cases are observed, which seem to occur without any obvious cause, especially in infants. It may here be mentioned that encysted hydroceles may suppurate, especially after puncture, and that suppuration may also occur in the sac of an ordinary hydrocele, but these cases must not be confounded with those of abscess of the testicle and epididymis.

There can be little doubt that so far as frequency is concerned, the commonest abscess formation of these parts is occasioned by the breaking down and softening of deposits of tubercle. All those who write upon the subject of tubercular disease of the testicle have noted the familiar pathological and clinical phenomena of suppuration. The commoner form of this kind of testicular abscess is the multiple. Here numerous irregular cavities are found scattered throughout the testis and epididymis, their walls lined by a distinct membrane, communicating with the exterior by sinuous fistulæ, containing 'curdy' pus, or inspissated masses of cheesy appearance and putty-like consistence. The more exceptional variety of tuberculous abscess of which I have seen several examples, is that in which the whole testicle is destroyed, being represented by a large abscess cavity containing yellow pus and limited by a thick leather-like membrane, often studded upon its inner surface with caseating deposits. Such an abscess reminds one forcibly of an old phthisical cavity in the lung, or the sac of a suppurating kidney in tuberculous pyelitis. In the Museum of St. George's Hospital is a typical specimen of this nature. By the kind permission of the Medical Committee I have had a drawing of it executed by Mr. Mark. The softening and disintegration of syphilitic gummata in the testicle is comparatively exceptional. In broken down cachetic individuals, suffering from severe attacks of syphilis, this condition is familar to practical surgeons. The abscess which ensues usually contains a more or less circumscribed slough of disintegrated tissue, which oftentimes is tedious in separating, after evacuation of the more fluid contents

of the abscess cavity. Though suppurations in connection with tubercle and syphilis are recognised and often met with in practice, they hardly deserve lengthy consideration in this paper, since the abscess formation is to be regarded as an occasional association of constant local pathological conditions. These cases, however, need special mention. In suppuration of the testicle due to other causes, the influence of a constitutional taint of tubercle or syphilis may play a very important part, both in the causation of the mischief, and in its treatment, but especially the latter.

Considering the extreme frequency of inflammation of the epididymis in the later stages of gonorrhœa, suppuration is extremely rare. It is, perhaps, not far wide of the truth to say that it never occurs in healthy individuals. Hunter mentions that he has known gonorrhœal epididymitis suppurate.[1] Robert[2] states his opinion that gonorrhœal epididymitis rarely terminates in suppuration, and when it does tubercles will always be found in the suppurating parts. Gosselin relates a case of sub-acute abscess of the epididymis in a man of fifty-nine following gonorrhœal epididymitis, which required opening by incision. Sir Astley Cooper also speaks of the rarity of this affection. It is otherwise with inflammations of the testicle and epididymis, the result of other irritations in the urethral canal, or about the prostate. Here abscess is occasionally observed.

The passage of a catheter during gleet may be followed by orchitis and suppuration. Mr. Harvey Ludlow, in his unpublished prize essay,[3] relates a typical case of this nature. An emaciated man aged fifty-eight had acute symptoms after catheterisation, as fever, rigors, agonising pain and swelling in the testis. The urine was putrid and offensive. The testicle was ultimately incised by Stanley, and a foul abscess, surrounded by gangrenous testicle

[1] 'Treatise on the Venereal Disease,' p. 218.

[2] 'Maladies Vénériennes,' p. 218.

[3] Library of the Royal College of Surgeons.

substance, evacuated. The whole gland was disorganised, and the patient subsequently died. The same author narrates the case of a delicate boy aged sixteen who was brought to St. Bartholomew's Hospital on the 3rd of February, 1831. A week before he had passed a piece of catgut into the urethra, and his symptoms dated from that time. The scrotum was swollen and œdematous. Incisions made by Stanley evacuated pus from the left scrotum and right testis. A favourable recovery ensued.

In the 'University College Hospital Reports'[1] the case is related of a man aged forty-two who for ten years had a catheter occasionally passed. The right testis had become swelled and painful for three weeks before admission. Finally, an abscess burst and discharged profusely. There was iritis in this case, and a fusiform tender swelling over the shaft of the left femur, which subsided under biniodide of mercury. The sinus in the testis was enlarged, and the cavity "mopped" out with solution of chloride of zinc.

A gentleman aged forty-four was a patient under the care of Mr. Buckston Browne, suffering from atony of the bladder and stricture. For three years catheterisation was occasionally resorted to. In January, 1890, this gentleman had "influenza" and this was followed by right orchitis. In February fluctuation was detected, and a foul sloughy abscess opened by incision. The whole gland was subsequently removed, as it was soft and disintegrated, and a favorable recovery ensued. When laid open the gland showed an abscess cavity extending some way into the substance of the testicle, and surrounded by flocculent, shreddy tissue.

Hutchinson relates the following cases:[2]—An old man, after the passage of a catheter, suffered from acute inflammation of the left testis. Fluctuation was detected, and an incision gave exit to turbid serum from the tunica. The testis was soft and congested.

[1] 1886, p. 70.

[2] 'Med. Times and Gazette,' 1871.

An old gentleman of eighty, in whom the right testis had passed into gangrene, and had been exposed by a sloughy abscess, had long suffered from prostatic irritation, and had passed calculi.

In the person of a gentleman aged thirty, who suffered from gleet, right acute orchitis supervened upon catheterisation. Some thick dirty pus was evacuated from the substance of the testicle itself.

Gosselin[1] relates two striking cases of orchitis and suppuration of the testicle, when the tunica vaginalis was filled with pus, and the gland quite disintegrated; one of these was in the person of an elderly man who suffered from prostatic irritation and dysuria. The other, seen with Brouardel, was in a lad aged sixteen, and attributed to masturbation.

I have not been able to find a single case of abscess of the testicle complicating the operation of urethrotomy. I have seen, however, severe orchitis associated with this proceeding.

Operation for stone, whether by crushing or perineal lithotomy, may be complicated by severe orchitis and abscess of the testis.

Terillon and Mounod state that Demarquay thrice experienced cases of abscess of the testis after lithotomy. In this country such a complication is exceedingly rare, if one may judge from the published reports of our principal operators. In India the case is different.

An article by Garden in the 'Medical Times and Gazette' for 1871 deals with this subject. He analyses the large number of 799 cases of lithotomy performed during twenty-three years commencing in 1848. The total number of cases of orchitis were sixteen. In five of these the testis suppurated, and incision was requisite for the evacuation of pus. He suggests that some kind of epidemic influence prevailed, for three cases occurred in each of the years 1849, 1854, and 1869. He found the aged more disposed to this complication than the young,

[1] 'Clinique Chirurgicale,' tom. iii, p. 389, *et seq.*

and that the left testis was more often affected. This he attributes to the position of the incision into the neck of the bladder, on the left side.

The operation of lithotrity is occasionally complicated with orchitis and abscess of the testicle. This was probably more common in the days of repeated crushings, and irritation of the urethra by fragments of stone, than at the present day. Thompson remarks that orchitis is not an infrequent sequel of lithotrity.[1] Hutchinson[2] relates the case of acute suppuration of the right tunica vaginalis after lithotrity. The testis escaped, but a succession of bad abscesses formed about the prostate, and in the iliac fossæ.

An old man was an inmate of St. Bartholomew's Hospital in 1855, and underwent several operations of crushing the stone at the hands of Stanley. After the fifth sitting in which the stone had been freely crushed, he had on the fourth day an attack of retention of urine, with all the symptoms indicative of the impaction of a calculous fragment in the urethra. Swelling of the right testis, penis, and scrotum at once supervened. There was no urinary extravasation. Subsequently an acute abscess formed, and this being opened exposed the gland in a sloughy condition. Endeavouring to remove the grey slough with the forceps, Stanley drew forth many yards of convulated tubuli seminiferi. The man recovered, but his testicle was destroyed.[3]

In connection with this case, may be mentioned a specimen in the Museum of St. Bartholomew's showing an abscess of the testicle after lithotrity. The gland is soft, enlarged, and appears to have been exceedingly congested.[4]

It seems that so far as orchitis and abscess after catherterisation are concerned, such cases are apt to present

[1] 'Diseases of the Urinary Organs,' p. 253.
[2] 'Med. Times and Gazette,' 1871.
[3] Ibid., Dec. 6th, 1856.
[4] 2760.

themselves in those who have long suffered from chronic irritation of the urethra, prolonged gleet, constant catherterisation, or prostatic irritations. It is usually agreed that the cause is the spread of inflammation along the seminal duct. In some cases, and especially after operations, a septic factor may be introduced and thus produce suppuration. Or this latter termination may occur in the aged, feeble, or intemperate, whose tissues are more prone to suppuration than healthy repair. I believe that cases of orchitis of this nature are more frequent than the published records of them would lead us to believe.

Injuries of the testicle itself producing blood extravasation are rare, suppuration as a consequence is still more exceptional. Experiments have been made in this direction by crushing the testes of animals. Though extensive blood extravasations were thus produced, abscess was not observed to follow.[1] In the south of England it was long the practice to emasculate animals by crushing the testes. Inflammation and atrophy resulted, suppuration was rare. Encysted abscesses of ancient date are sometimes found in the testis, and it is difficult to explain their occurrence unless on the supposition of old injury. Such are to be distinguished from the scrofulous by the absence of cheesy products in the interior, or of caseating inflammation in the surrounding testicular tissues. There are two specimens of encysted abscess of the epididymis in the Museum of St. Thomas's Hospital.[2] Another exists in the Museum of University College. The Curator, Mr. Stonham, describes it as a chronic abscess without doubt, but there is nothing in the specimen to enable one to assign an exact cause for its occurrence.[3] In the London Hospital reports, the case is related of a man aged sixty-nine, in whom a swelling of one testis had existed for twelve months; hydrocele was likewise present. The

[1] 'Archiv Gén. de Méd.,' 1881, tom. ii.

[2] St. Thomas's Hosp. Museum, E. E., 16, 17.

[3] 'University Coll. Cat.,' p. 418.

testicle when cut into contained an abscess in its interior.[1] A case of chronic abscess of the testis is tabulated in the Surgical Registrar's reports of University College Hospital for 1882. In the Surgical Reports of the same hospital two cases are narrated bearing upon this part of the subject. A man aged fifty, after an injury, got acute pain and swelling in the right testicle with sickness. Leeches were applied without benefit. On the thirteenth day an abscess was detected, and opened by trocar and cannula.

A man aged fifty-eight had repeated abscess of the testicle for twenty-five years, after a kick from a horse. There was no syphilitic history. It was thought advisable to remove the whole mass. When this was done an abscess cavity was found in each testicle.[2] An abscess following a blow, with fungation of the organ, is also related by Rémy.[3] Gaussail places on record a case of orchitis following the kick of a horse, when multiple abscesses formed in the testicle, and the whole organ was destroyed.[4] A time honoured authority, Sir Astley Cooper, speaks especially upon abscess of the testicle, connected with injury. He tells us that the abscess generally breaks at several apertures and sinuses follow, which are very difficult to heal, for they issue a seminal as well as a purulent discharge.[5] It may be pointed out in leaving this part of the subject, that it is very needful to distinguish between abscess of the cellular tissue about the scrotum or epididymis, and purulent collections in the substance of the testicle itself. Abscess in the scrotum is found from a variety of causes, among which may especially be mentioned old stricture and extravasation of urine, and necrosis of the rami of the pubes, or its symphysis.

In general pyæmia, abscess may occur in the testicles.

[1] 'London Hospital Reports,' 1875-77, p. 64.

[2] 'University College Hospital Reports,' 1887, p. 78.

[3] Rémy, 'Journal de l'Anat. et de la Phys.,' 1880.

[4] Gaussail, 'Archives Générales de Méd.,' 1831, vol. xxvii.

[5] 'Treatise on Diseases of the Testicle,' p. 11.

Wilks and Moxon, in their work on pathology, state that they have four times found abscesses in the epididymis in cases of pyæmia. There is a specimen in the Museum of St. George's Hospital to illustrate this affection. It is the right testis of a man aged twenty-eight, who was admitted into the medical wards for supposed acute rheumatism, affecting the left shoulder. Swelling and pain in the scrotum supervening, he was transferred to the care of Mr. Tatum and died in August, 1851. The description of the specimen in its recent state tells us that the organ was congested and soft, and in its substance were deposited several masses and spots of pus. Subcutaneous abscesses and pustules on the skin were noted with secondary abscesses in the right lung. Lapse of time and prolonged maceration have destroyed the morbid appearances above described. In the Museum of Guy's Hospital is a specimen of interest—the testes of a man aged thirty who died of general pyæmia, with suppuration of the cerebral sinuses, lungs, and joints. In the right testis the epididymis is greatly enlarged and congested, but near the globus minor and in the testis are some caseating spots, which may well cause a suspicion of tubercular disease. The left testis is engorged and enlarged, and the epididymis swollen to thrice its natural size. That this was the result of pyæmic inflammation seems very probable, and such was the opinion of the pathologist who described and mounted the specimen.[1]

Abscess in the testicle or epididymis as a sequel or complication of the specific fevers is a matter of great interest, but seems to have attracted no notice in this country. Murchison, in his classical treatise on the continued fevers, does not allude to it, neither is it referred to in the leading text-books of medicine. I have written to the Medical Superintendents of each of the large Fever Hospitals in Great Britain and Ireland on this subject. They have been courteous enough to grant me full information, but I have for the most part received negative replies. Several of them have the candour to state that their atten-

[1] Museum and Catalogue of Guy's Hospital, 2341[50].

tion has never before been drawn to the subject, and that cases may have been overlooked. This is sufficient evidence of the rarity of this complication of fevers in this country.

Trousseau[1] notices the communication of Béraud on "Variolous Orchitis," and states that since he (Béraud) wrote, many cases have been observed, not because they were more common now, but because we look out for them. In Béraud's article,[2] he notes that the orchitis of small-pox is bilateral, and accompanies the skin eruption. He describes epididymitis and pustular inflammation of the parietal layer of the tunica vaginalis.

The usual termination was in resolution, and the so-called "critical" abscesses that occurred occasionally in the scrotum were really suppurations in the cellular tissue near the tail of the epididymis. Lancereaux, in his 'Atlas de l'anatomie pathologique,' gives a good illustration of multiple abscess in the testis of variolous orchitis.[3]

It is, however, especially after typhoid fever that abscess of the testicle occurs, though the fact is not noted by those of our leading pathologists who have written upon the Surgical Sequelæ of the eruptive fevers. Terillon and Mounod speak very definitely upon this point.[4] "Typhoid orchitis more often terminates in abscess than any other variety of orchitis which causes testicular abscess. . . . The abscess is acute and rapid, tending speedily to invade the scrotum, the testis is completely destroyed." One of the earliest recorded cases of this nature is by Hanot. He relates the case of a young man, free from tuberculous or syphilitic disease, who, subsequently to a severe attack of typhoid fever, suffered from right orchitis and suppuration of the testis, with complete extrusion of the gland through the lancet puncture.

[1] 'Clinical Med.,' ii, p. 57.

[2] "Recherches sur l'orchite et l'ovarite varioleuse," 'Arch. Gén. de Méd.,' tom. xiii.

[3] Plate 37, p. 365.

[4] Terillon et Mounod, p. 374.

In another article he refers to similar cases.[1] Liebermeister notes the occasional occurrence of orchitis during convalescence from typhoid, but in his experience the cases are mild.[2] In the 'Lancet' for January 22nd, 1881, there is an article noticing orchitis as a sequel of the so-called "Mediterranean fever." The author remarks upon the pain and swelling of the joints which accompanied these cases, and asks whether the orchitis is rheumatic or otherwise. With regard to the true nature of this fever, Paget remarks—"The fever which I have referred to as closely related to typhoid, is that which in the last two or three years has been prevalent in Naples, Rome, and I believe other places in Southern Italy."[3] There is a most interesting article by Duffey in the 'Dublin Journal of Medical Science' upon this subject.[4] After attacks of fever considered to be enteric, orchitis was frequently noted as a sequel among the men of the 24th regiment. Eighteen cases are mentioned. The attack was sudden, violent, painful, the scrotum on the affected side being distended to the size of one's fist. These cases, also, were accompanied by pains in the joints and fascial structures; hence the term 'rheumatic,' was applied to them. In one case, where a delicate young man was much prostrated by a severe attack of fever, the orchitis terminated in suppuration with protrusion and fungation of the testicle, but a good recovery ensued. This writer refers to another case in the same epidemic which terminated in suppuration. It is worthy of note that numbers of cases have occurred together, frequently among troops, where a large number of individuals are closely associated.

In the Museum of Charing Cross Hospital is a right testis, enlarged and congested, with a large, sloughy abscess cavity in its interior. This was removed by Mr.

[1] Hanot, 'Soc. Anat. de Paris,' 1873, vol. viii, p. 589; 'Archives Gén. de Méd.,' Nov., 1878, p. 595.

[2] Ziemssen, 'Cyclopædia,' p. 181, vol. i.

[3] 'St. Barth. Hosp. Reports,' p. 2, vol. xii.

[4] 'Dub. Journal of Med. Science,' Feb. 1st, 1872.

Bloxam from a young man, who remained pale, ill, and exhausted, about six weeks after apparent convalescence from typhoid. The scrotum had continued to enlarge slowly, since the third week of the fever, and the characters of abscess were by no means defined. On incision, the *tunica vaginalis* was full of pus, which, proceeding from the sloughy abscess of the testicle, led to the removal of the latter organ, and a satisfactory recovery. For a knowledge of the following case I am indebted to Mr. Hopwood, the resident Medical Officer of the London Fever Hospital. A severe case of typhoid in a lad aged nineteen was in the hospital in the year 1879 during the physiciancy of Dr. Broadbent. He was treated with cold baths and quinine. After passing through much peril it was noted about the thirtieth day of the disease that there were numerous pustular abscesses over the face, shoulder, chest, and back. On the fortieth day a collection of pus was evacuated from the right scrotum, and it was noted that the left testicle was swollen and tender, with an indistinct sense of fluctuation. Subsequent incision evacuated pus, with much discharge of sloughy tissue from the testicle itself.

A like case has occurred to Dr. David Macleod of Glasgow, to whom I am indebted for the particulars. A man, aged twenty-five, was admitted on the sixth day of enteric fever to the Glasgow Fever Hospital, suffering from a severe attack. On the forty-sixth day the right testicle was noticed to be greatly swollen and very tender. On the fifty-ninth day the organ was incised, and a quantity of foul pus evacuated. The man soon became convalescent, and the pyrexia, associated with the testicular affection, which was long believed to mark a "relapse" of the fever, disappeared. The same physician informs me that in another of his cases severe orchitis occurred, and suppuration urgently threatened. Dr. Sweeting of the Local Government Board, whose experience of the eruptive fevers has been large, has met with one case of severe orchitis after typhoid, when suppuration was averted by

early elevation of the inflamed organ, and other measures. He sends the particulars of a case of suppuration of the testicle after scarlet fever. This happened in a boy, aged six, who was admitted on June 2nd, 1886, to the Fulham Fever Hospital. The lad was desquamating, with cervical adenitis, nephritis, and dropsy. Subsequently a fluctuating swelling was detected in the scrotum which was opened on 19th July. Much pus escaped, apparently from deep down; the testicle was enlarged and inflamed. Sloughing and gangrene of the genitals is known after typhus, but I have been unable to hit upon a distinct case of abscess as a complication of this fever.

As regards the explanation of orchitis and abscess after typhoid, one can hardly doubt but that this complication is of a pyæmic nature. The pains and swellings of the joints noted by some observers are suggestive. Indeed one would rather class these cases of inflammation and suppuration with the surgical sequelæ of the eruptive fevers, as abscesses of joints, necrosis, and the like. These are usually looked upon as of a pyæmic nature, occurring in feeble individuals, whose health generally is shattered and undermined by the prolonged ravages of an acute and dangerous malady.

This subject would hardly be complete without reference to the close association between 'Mumps' and orchitis. Mumps is more often followed by orchitis than any of the specific acute diseases. Though the inflammation is often exceedingly acute, and associated with the most alarming constitutional symptoms, I am unable to find records of a single case of suppuration. Velpeau, in an article on the orchitis of 'mumps,' does not mention suppuration.[1] Laurens writes on an epidemic of mumps and orchitis in the French Army. Atrophy of the testicle occurred, but without suppuration.[2] Laveran relates 156 cases of orchitis after mumps, without suppuration.[3]

[1] 'Brit. and For. Med.-Chir. Review,' 1857, p. 271.

[2] 'Med. Times and Gazette,' 1877, Feb. 24th.

[3] Laveran, 'Med. Times and Gazette,' 1878, July 20th.

The same fact is noted by Pilliet and Rèssiguier, in their account of an epidemic of mumps at Geneva in 1848.[1] I have written on this point to the medical officers of the large public schools of this country. They all have had experience of the orchitis of mumps, but have never seen a case of suppuration. Dr. Dukes, of Rugby, has published twelve cases of orchitis as a sequence of mumps, in one of these acute hydrocele was present, and paracentesis had to be resorted to, but no suppuration occurred.[2] It is interesting to note the similarity in behavior between the testicle and parotid gland in this respect. Suppuration of the parotid gland in mumps is almost unknown, yet parotid abscess is a known complication of pyæmia and of the eruptive fevers.

Abscess may occur in the testicle of infants. When acting as resident obstetrician to St. George's Hospital I observed a case of this nature. A puny infant, the offspring of poor and dirty parents, suffered from a succession of abscesses in the left axilla, apparently connected with ulceration of the umbilical aperture. The left testicle became red, hot, swelled, and painful. Finally, it was incised with a lancet, about a drachm of pus escaped, but the gland was entirely destroyed. A case is related in the 'Medical Times and Gazette'[3] of a child six weeks old, who was brought to Mr. Bryant with an abscess of the right testicle. The abscess had existed for a week, and had burst before admission. The testicle was much enlarged, and the resulting sinus passed deeply into the gland.

As regards the treatment of abscess of these parts, it appears to be of paramount importance that an early opening shonld be made, especially in acute cases. The large amount of sloughing and disintegration of the secreting structure of the testicle, with the great pain and agony, are doubtless due to the action of acute inflam-

[1] 'Brit. and For. Med.-Chir. Review,' 1850, p. 541.

[2] 'Lancet,' Oct. 29th, 1881.

[3] March 22nd, 1862.

mation within unyielding tissues. The question of treating acute inflammation of the testicle by incision has been much disputed, but still has its advocates. In cases where suppuration threatens a moderate incision into the enlarged gland, made with a lancet under anæsthesia, may, by the free abstraction of blood, and relief of tension, obviate this very serious termination of the inflammatory process. When the abscess is burst or has been evacuated, and a resulting sinus, or fungating testicle substance remains, the question of removal of the entire gland will arise. It is here that the presence or absence of a tubercular element in the case is especially important. Should the surgeon be able to assure himself of the absence of tubercle, it is well not to be in an extreme hurry to perform the radical operation. Much of the fungating and disintegrated tissue is of inflammatory formation, rather than of true testicle structure, and subsequent healing may take place, through patient treatment, with the preservation of the major part of the gland in an unimpaired condition.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 14.)

ON 114 CONSECUTIVE CASES

OF

OPERATION FOR CALCULUS IN THE BLADDER,

PERFORMED AT THE HOSPITAL FOR SICK CHILDREN,

AND A COMPARISON OF THE METHODS ADOPTED.

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THERE are recorded in the accompanying table 114 cases of calculus in children below the age of twelve. The methods and results of removal are as follows :

		Recovery in	Deaths.
By lateral lithotomy . . .	75 ...	71 ...	4
By vesico-vaginal lithotomy . .	9 ...	9 ...	0
Calculus in urethra . . .	4 ...	4 ...	0
By lithotrity	11 (3 F.) ...	10 ...	1
By supra-pubic incision . .	15 ...	13 ...	2
Total . . .	114 ...	107 ...	7

Dealing first with lateral lithotomy we have 75 cases with 71 recoveries, giving a percentage of 94·6. Comparing this with the tables collected by Sir H. Thompson,

we find that out of 850 boys operated on by the lateral method between the ages of one and twelve, that is to say the same as are included in the accompanying tables, there were 811 recoveries, or a percentage of 95·41. The statistics of Mr. Charles Williams of the Norfolk and Norwich Hospital give the results of 335 operations in boys between 1 and 10 years, and out of this number there recovered 314, giving a percentage of 93·73. It is more than probable that the former include the latter, but the tables are useful as showing the almost identical results of the operation under various circumstances.

Of the four *deaths* which occurred after the lateral operation one (No. 41) a child, æt. 8, had had stone in the bladder for about seven years, and died from old abscess in the kidney. A second boy, æt. 2, was very feeble (No. 48), and died of pleurisy and diarrhœa. The third case (No. 20), æt. 8, died on the twenty-second day after the operation from scarlatina, which appeared two days after the lithotomy. The fourth death (No. 54) was due to pyæmia.

Another boy, æt. 2, recovered from the operation but died three weeks later from croup, for which tracheotomy was performed. This case is not included among the deaths.

Hæmorrhage.—Of the *casualties* occurring after the operation hæmorrhage took place in six instances (Nos. 3, 6, 25, 28, 29, 92). In one (No. 3) arterial hæmorrhage from the deep part of the wound recurred up to ten days after the operation. Plugging round a tube was kept up for fourteen days, after which the wound rapidly closed. Plugging was required in a second case (No. 6). Two attacks occurred in another child (No. 25), and it took place on the second day (No. 28) in the fourth case, and (No. 29) one child had hæmorrhage followed by an abscess in perineo and testis. In the last case (No. 92) there was considerable hæmorrhage after operation, and a little more on the eighth day. All these patients recovered.

Abscess.—Abscess also occurred in the left side of the

scrotum in No. 1, and in No. 2 there was abscess in the left testis, pus in the urine and pain in micturition for some time, but ultimately a perfect recovery.

Erysipelas followed by scarlet fever and measles took place in one patient who also recovered (No. 26), and *scarlet fever* followed by albuminuria occurred in another instance which also recovered (No. 31). No. 33 got scarlet fever two days after coming into hospital. After operation a great deal of pus in urine, and albuminuria. No. 87 scarlet fever and ultimate recovery.

In one patient, æt. 5 (No. 78), much trouble followed the operation from atony of the bowel and accumulations of fæces, but the recovery was satisfactory, although three stones were removed at the time of operation. Most of these casualties occurred before the opening of the new hospital in the autumn of 1875, and some may therefore be fairly attributed to the faulty hygienic surroundings of the patients in the building which has now disappeared.

One boy had orchitis on the eighteenth day after removal of a very large stone (No. 61). One patient (No. 110), æt. 3 years 2 months, whose wound remained open for some weeks, caught nasal diphtheria and was in separate ward for five weeks, at the end of which the sinus had closed.

Of the size and nature of calculi.—Of the calculi removed by the lateral operation, some were remarkable for their size, shape or other peculiarities. In two cases (No. 23, Spec. G.G. 30; No. 78, Spec. G.G. 81)[1] three calculi were removed from the bladder of boys æt. 8 and 7. In four instances two stones were found (No. 49, 67, Specs. G.G. 45, 62; Nos. 71, 73, Specs. G.G., 67, 70) in boys of the ages of 2 years 6 months, 6 years 11 months, 2 years 3 months, and 2 years 4 months. The weights in the latter case were 42 gr. 44 gr. respectively. A calculus of peculiar shape and resembling two oval stones united by a narrow band, which weighed 63 gr., was

[1] The specimens are to be seen in the museum of the hospital, and are described in the catalogue prepared by Mr. Morgan, to which the numbers refer.

removed from a boy æt. 6 (No. 17, Spec. G.G. 19). A uric acid calculus encrusted with phosphatic material and weighing 229 gr. was taken from a boy æt. 2 years 6 months (No. 45, Spec. G.G. 41), and a uric acid stone of 178 gr. from a boy 5 years 5 months (No. 60, Spec. G.G. 55), and a calculus with uric acid nucleus of 450 gr. from a boy æt. 11 (No. 57, Spec. G.G. 52). Other stones of uric acid and weighing respectively 64 gr., 151 gr., 108 gr., 140 gr., were found in boys aged 8, 6, 2, 2, 11, 9 (Nos. 61, 63, 64, 66, Specs. G.G. 56, 58, 59, 61). Calculi of oxalate of lime existed in two instances (Nos. 58, 65, Specs. G.G., 53, 60). They weighed 53 gr. from a boy æt. 9 years 6 months, and 36 gr. from a patient, æt. 4.

With regard to the recurrence of calculus; one child, a boy, æt. 11, was twice submitted to the lateral operation at an interval of five months (see Nos. 29 and 32). On the first occasion April 19th, 1871, there was hæmorrhage followed by abscess in perineo and testis, and the wound had not closed on September 23rd, when another small stone was removed. This was followed by great pain in both hips and some slight peritonitis, but the patient ultimately recovered.

In one instance (No. 37) a boy, æt. 8, had been sounded without result at St. Bartholomew's Hospital three months previous to his admission at Great Ormond Street, when he came under the care of the same surgeon, who removed a stone, and another boy had passed a calculus in the hospital the night before he was to have been operated on. He was, however, carefully sounded with no result. Ten months later he was re-admitted and a stone weighing 63 gr. was removed. And an instance of recurrence was found in a boy, æt. 11 (No. 52), who had undergone the lateral operation for a stone the size of a pea at another place thirteen months before admission, when a second stone of uric acid and weighing 88 gr. was removed by the same method.

One specimen of cystine calculus (Spec. G.G. 47) was taken from a boy, æt. 9 years.

It is noteworthy that there was no single death in all the cases of large or peculiar stones.

The four cases in which a calculus was found in the urethra call for no comment. All were removed, either by forceps introduced into the urethra or by a small incision, and were followed by no complication.

Females.—Of the total number of cases of calculus (114) twelve occurred in female children and of these eight were treated by vaginal lithotomy, one was removed *per urethram,* and three by the crushing operation. In all cases but two the opening in the vesico-vaginal wall closed and left no fistula, but in one case (No. 31) a uric acid calculus coated with large quantities of phosphates, which caused difficulty in its removal was successfully extracted, and the edges of the incision were closed with five sutures, but a fistula resulted, and there is no record of its having been finally closed (No. 36). Another patient had a fistula which was closed subsequently.

Lithotrity.—Of the three cases in females which were subjected to lithotrity one was submitted to two operations at a week's interval, but all were completely and rapidly cured. Eight male patients were treated by lithotrity, the youngest being two years and eight months and the oldest five, the eight giving an average age of three years and eleven months (Nos. 105, 111). In one boy the operation was performed on two occasions with a year's interval. All others were successfully treated at one sitting. The weight of the stone was 43½ grs. in a boy (No. 96), æt. 3 years 3 months, the whole being evacuated at the time of operation except one small fragment which passed naturally two days subsequent. Fifty-four grains of uric acid calculus were crushed and washed out from the bladder of a boy, æt. 3 years 6 months (No. 100); a very little gravel was passed on the two subsequent days, but on the fifth the boy was up and about in the ward.

The one fatal case occurred directly from the operation. The patient was an unhealthy looking boy, æt. 5, and a stone had been successfully crushed when the bladder

was injected with water from the evacuator. Suddenly this viscus which was distended with water was seen to collapse, and laparotomy was immediately performed, when a rent was found in the anterior wall at a part which did not involve the peritoneum. This was sewn up and a perineal cystotomy performed in order to prevent the escape of urine into the abdominal cavity, the patient, however, did not recover from the collapse and died in the course of a few hours.

Fifteen cases, all males, were treated by the suprapubic operation as modified in recent years, the bladder being washed out and afterwards distended by injection of boracic lotion, and an inflated bag of india-rubber inserted in the rectum. The first operation in this manner was performed on July 29th, 1885. Of these, thirteen recovered and two died. The death in one case, a patient, æt. 4 years and 2 months, was brought about by the boy having suffered from diarrhœa which was not reported by the nurses. The operation was performed in the usual manner, a moderate sized stone extracted and the incision in the bladder was united with catgut. The skin wound was also united and a drain inserted at the lower margin. Urine issued from the wound on the day of the operation, but the boy became collapsed and died on the third day following. Peritonitis was found at the examination, but the peritoneum had not been injured. In this case an attempt had been made on a previous day to pass a lithotrite, but was abandoned on account of the smallness of the urethra (No. 103).

The second death occurred in a boy æt. 9, in whom also an attempt to use the lithotrite was made but had to be abandoned on account of the size of the stone. The usual operation was performed and a large stone extracted (No. 109). There was a good deal of phosphatic deposit which required to be washed out of the bladder. The incision was not stitched. In the evening hæmorrhage occurred and two vessels required to be ligatured. The boy died in the evening of the following day, and as no

satisfactory cause could be ascertained it was presumably due to septicæmia.

Of the casualties that followed this operation one patient after recovering from the operation had some nasal discharge and was afterwards affected with diphtheritic paralysis which placed his life in jeopardy for some time.

In No. 89 there was slight extravasation of urine after vomiting and much urine passed through the wound, which, however, healed on the thirteenth day. In a case (No. 91) where the muscular coat of the bladder was united by catgut sutures, and the incision in the abdominal wall by silk, a catheter was left in the bladder to drain it *per urethram*, but this causing urethritis was withdrawn, and the bladder became distended with urine so that the stitches were torn out and some hæmorrhage took place into the viscus, but on the clots being washed out the boy eventually made an excellent recovery (*vide* 'Lancet,' May 7th, 1887).

In a boy (No. 95) æt. 1 year and 4 months, weakly and rachitic, symptoms of calculus had existed for three months and a stone was found on sounding which did not readily move in the bladder, and when the suprapubic opening was made a stone was felt lying in a sacculus in the region of the trigone, below and between the orifices of the ureters and overlapped by the mucous membrane of the bladder to such an extent that it became necessary to raise the calculus from below by the assistance of the forefinger in the rectum. The stone was of uric acid weighing fifty-three grains and was of the shape of a blunted cone, the apex of which was buried in the walls of the bladder. No sutures were used and the wound healed on the sixth day (*vide* 'Lancet,' October 22nd, 1887).

In another case a calculus was found to be fixed in the mouth of the ureter (No. 97).

Of two cases in which the bladder wound was sutured with carbolised catgut, in the first (No. 101) urine escaped through the wound on the fourth day but ceased to do so

at the end of fourteen days, and union of the wound was complete at the end of a month. In the second (No. 102) the incision of the bladder united at once, but suppuration occurred in the superficial parts which delayed union till the end of three weeks, and in a third (No. 104) instance which was similarly treated urine leaked through the wound on the third day but ceased on the seventh, and union was sound on the seventeenth day.

In reviewing the results of lateral lithotomy it is impossible not to be struck with the very great success which has attended its performance. Of the deaths which occurred in patients who were subjected to this form of operation only one out of a total of seventy-five cases can be attributed as directly due to the operation, namely the case (No. 54) of a boy, æt. 2 years, who died of pyæmia. It is true that this occurred in the wards of the new building, but it was in the year 1877 when precautions against sepsis were not universally carried out with that punctilious observance which is now-a-days almost invariable, and it is possible that even this death might have been prevented with more rigid precautions. Of the other deaths two were no doubt due to the changes in the kidneys resulting from the continued presence of stone, and the other was the result of epidemic diseases which must prevail at intervals in all institutions where a large number of children are congregated. Hæmorrhage was the most frequent and the most serious of the casualties following the operation, but death did not occur in a single instance in which hæmorrhage took place, nor was there a fatal result in any of those cases where abscess, orchitis, erysipelas, &c., which are among the recognised sequelæ of this operation occurred, and the number of instances in which any of these sequelæ happened is remarkably small out of so large a number, seeing that the patients were from among the very poor and ill-fed children, and were suffering from a disease which is largely engendered by ill-advised feeding from a very early age, as well perhaps as from a faulty constitutional

state. Due credit must also be given to this operation for the success which has attended the removal of even the largest stones, the size of which has in many instances been mentioned, whilst from the specimens before the Society it may be judged that there are many instances, not mentioned above, where the calculus must have presented difficulty in extraction either on account of its shape, or size, or other peculiarities.

Although, in one instance, the urethra was torn and the stone was not extracted at the time of the operation, it came away through the wound on the following day and the patient ultimately did well. It should be noted that there is no single instance amongst these seventy-five cases in which the rectum was wounded, notwithstanding the large size of some of the calculi removed, and due credit will be given for this by those who have had occasion to treat patients in whom this untoward accident has taken place.

It is, of course, impossible to say whether any ulterior consequences have supervened in any of these cases, such, for instance, as have been pointed out as liable to result from this operation by reason of injury to the vesiculæ seminales. There are obvious reasons why such results can never be ascertained as following an operation performed on children many years before the advent of puberty. But the immediate casualties resulting from the operation are sufficiently numerous, notwithstanding their immunity from fatal consequences, to make it desirable to avoid a cutting operation, even under the improved methods now in vogue, the origin of which is subsequent to the period in which a large proportion of these cases were treated.

On the other hand, a glance at the collection of stones here exhibited makes it evident that in a large number of instances the surgeon would have preferred to avoid an operation involving risk either immediate or secondary, if the removal of the calculus could with equal efficiency be brought about by other means, and if the bladder could be thoroughly relieved of all its foreign contents without

danger of any remaining to form the nucleus of a fresh concretion. The list of cases which have been treated by lithotrity is but a small proportion of the whole number, but the results have been most satisfactory, with the one exception of death occurring as an immediate accident of the operation. In all the other cases the stone was crushed and completely evacuated at one sitting, except in the case of a girl (No. 81) who was submitted to two operations within a week. Of the eight boys in whom this operation was performed, except for the passage of small pieces of *débris* on the days following the operation, there has not been one who might not have left the hospital in the course of three or four days, but in those five upon whom I operated their discharge was delayed in order that I might feel certain that no fragments were left behind, and for this purpose the bladder was aspirated again at the end of a week, but in no single case were any more fragments washed out.

It will be noticed in the table that accompanies this paper that in three cases which were operated on, one by the lateral (No. 110), and two by the supra-pubic method (Nos. 103 and 109) the lithotrite could not be used. In two of these cases there was difficulty as to its introduction, and in one the stone was too large to be grasped by the lithotrite. But since the date of these operations Messrs. Weiss, from whom these instruments were obtained, have succeeded in producing efficient lithotrites of a smaller calibre. These are now to be obtained of the following dimensions :—

No. 5	...	5 in. stem	...	6 in. angle	...	opening	½	in.
6	...	6	„	...	7	„	...	„	½	
7	...	7	„	...	8	„	...	„	⅝	
8	...	8	„	...	10	„	...	„	1	

All these instruments are fenestrated.

The objections which have been raised against this form of operation in the case of boys have been so recently combated by Surgeon Major Keegan that it is unnecessary to repeat his arguments, and it will be generally admitted

that instruments of the size above-mentioned can be introduced through the urethra into the bladder of most boys over two years of age when the meatus has been incised, a proceeding which is hardly attended even by temporary inconvenience. Occasionally it happens as in two of the instances above mentioned, that, owing probably to a puckering of the mucous membrane of the urethra, in front of the projection of the end of the male blade, an impediment may occur to the introduction of the instrument, but this is the only circumstance which is likely to prevent its introduction to the bladder. As regards the capacity of that viscus, my own observation, drawn from a very large number of soundings of children who have been the subjects of stone, or have presented all the subjective symptoms of that affection, points to the fact that, owing probably to frequent distension, the bladder in these patients is exceedingly ample, and affords considerable space for the movements of a sound, or, if necessary, of a lithotrite, without danger of injuring the mucous membrane if ordinary care is observed. This being the case, I confess myself unable to see any objection to this form of operation in the case of stones of a small size, such as form a very large majority of those before the Society. Where the calculus is of such material, *e. g.* oxalate of lime, that its fracture cannot be accomplished by instruments as slender as those must be which have the above stated dimensions, the introduction of a lithotrite cannot prejudice subsequent proceeding, and the same may be said of the case of stones of such a size that they cannot satisfactorily be seized within the blades of the instrument.

Females.—As the records include only three cases in which lithotrity has been practised upon female children, the opportunity for comparing it with other methods is hardly sufficient, but again referring to the experience of those who have met with cases in which a vesico-vaginal fistula has resulted from the operation of lithotomy, it may fairly be urged that the former operation is to be pre-

ferred to one which runs the risk of causing this exceedingly intractable malady. It needs no arguments to urge the preference which should be given to the former operation in cases where the stone is of moderate size, or of a material which can easily be crushed. Not only is the above casualty avoided, but even the amount of dilatation of the urethra need be so slight as to cause no fear of its regaining its normal contractile powers. Very large stones, and others which do not lend themselves to the crushing operation, require to be dealt with by other means.

The subject of recurrence of calculus as a result of a fragment being left behind, I cannot but consider to be exaggerated as to its frequency, and as to its importance. Four out of five patients upon whom I personally operated, I have seen at intervals of two or three years, and in none has there been any evidence of symptoms of recurrence. On the other hand, a boy, who was treated by lithotrity by one of my colleagues in 1888, was re-admitted exactly a year later and submitted to the same operation (Nos. 105 and 111). Whether this stone in the second instance, which was judged to be of the size of a small nut, was a fresh one which had passed down from the kidney, or an accumulation around a fragment which remained in the bladder after the first operation, it is impossible to say, but I have seen the boy at a year's interval after the last operation and he has had no further symptoms of stone.

The writings of Surgeon Major Keegan have brought the subject of litholapaxy in the case of boys so forcibly before the profession that it is unnecessary to repeat the arguments which he urges in favour of its adoption by English surgeons. The brilliant series of results which he and several of his fellow labourers have obtained in India serve to encourage us in this country to follow their example. The main objections which have been urged against it are :—1st, The smallness of the urethra in boys and its extreme sensitiveness. 2ndly, The liability to recurrence which there may be if fragments are not

thoroughly evacuated. The first objection is shown by Surgeon Major Keegan to be exaggerated. With instruments such as are now manufactured, the urethra can be easily traversed, except in occasional cases where, as already narrated, the mucous membrane puckers in front of the male blade and causes difficulty to its introduction. But with improved instruments even this difficulty is not likely to occur, and as regards the sensibility of the urethra there is no reason why this should be greater in English children than it is in those born in India, and with the aid of an anæsthetic there can be no reason to avoid the operation on this ground. With regard to recurrence, it is, as has already been said, almost impossible to speak from facts, but while an instance has been given in which lithotrity has had to be repeated within a twelvemonth, another instance is mentioned where lithotomy was twice performed on the same patient within five months, and a second in which it was repeated after thirteen months, and in another boy a stone was removed ten months after another had been passed per urethram, and the probability seems that in one case as in the other fresh calculi passed down from the kidney, and as lithotrity is the operation from which recovery is most rapid and which leads to less complications, it seems indicated as the most desirable in cases where the stone is suited for the operation. It may be permitted me to say that in thus recommending the operation, I may endorse what is said by Mr. Keegan, viz. that lithotrity in the case of children is even a more delicate operation than in adults, and should not be attempted by any but those who have mastered all the manipulative details.

A safeguard against recurrence has already been mentioned, which is at the same time free from risk, and possibly even under all circumstances advisable, namely the washing out of the bladder by means of an evacuator, on a second occasion, at an interval of a few days. By this means not only is any remaining detritus removed but any fragment of such magnitude as to be unable to pass through

the urethra can be detected even more certainly than by the sound, and if discovered, can be treated with the lithotrite.

The author in omitting to refer to the excellent work which has been done in the direction of ascertaining the best means to be adopted in the removal of calculi from the bladders of the young of either sex has no idea of ignoring the admirable advice and conclusions which have been furnished in recent papers, more particularly by Mr. Jacobson and Mr. Walsham and others, but as the statistics herewith given form a history in themselves, the author has abstained from broadening the scope of the paper into a general treatise.

In conclusion I venture to submit that the facts disclosed in the accompanying table warrant the following propositions:

1. That in the cases of boys and girls small stones should be dealt with by litholapaxy.

2. That stones composed of oxalate of lime or of such size as not to be readily grasped between the blades of a lithotrite should be removed by the lateral operation in the case of boys.

3. That the supra-pubic operation should be reserved for stones of very large size or inconvenient shape in boys or girls, or cases of calculus embedded in a saccule of the bladder or impacted in the mouth of a ureter.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 18.)

Appendix to Mr. J. H. Morgan's Paper on Calculus.

No.	Date.	Sex.	Age.	Nature of operation.	Operator.	Result.	Remarks.
	1864.						
1	March	M.	6	Lateral	Mr. Holmes	Good recovery	Abscess formed in left side of scrotum, but was insignificant.
2	,,	M.	4	,,	Mr. Smith	,,	Very rickety child. Abscess left testicle. Pus in urine, and pain on micturition for some time after. Ultimately quite recovered.
3	June	M.	4	,,	,,	Recovery	Hæmorrhage, arterial, recurring up to 10 days after operation from deep part of wound. Plugging round a tube for 14 days. Rapid closure of wound.
4	July	M.	4.6	,,	Mr. Holmes	Rapid recovery	—
	1865.						
5	April 27th	F.	5	Dilatation and incision of urethra and vagina	Mr. Smith	Recovery May 7th	—
6	May 8th	M.	3	Lateral	,,	,, July 5th	Hæmorrhage after operation, requiring plugging of wound.
7	June 14th	M.	2	,,	Mr. Holmes	,, ,, 11th	Died after tracheotomy, July 26th, for croup.
8	,, 28th	M.	3	,,	,,	,, ,, 18th	—
9	Oct. 21st	F.	7	Lateral (?) lithotomy not stated	Mr. Smith	,, Nov. 6th	—
	1866.						
10	March 14th	M.	5	Lateral	Mr. Holmes	,, April 11th	—
11	April 18th	M.	10	,,	,,	,, May 20th	—

No.	Date.	Sex.	Age.	Nature of operation.	Operator.	Result.	Remarks.
12	May 23rd	M.	3	Lateral	Mr. Holmes	Recovery July 5th	—
13	Nov. 12th	M.	2.6	,,	Mr. Smith	Recovery	—
	1867.						
14	May 11th	M.	4.6	Calculus in urethra	Mr. Holmes	,,	Removal.
15	Oct. 19th	M.	8	Lateral (?) lithotomy (?)	Mr. Smith	,,	—
16	,, 26th	M.	3	Lateral (?) lithotomy	,,	,,	—
	1868.						
17	May 4th	M.	6	,,	,,	,,	—
18	June 6th	M.	3.6	Lateral	,,	,,	Weight 63 grs. Shape of stone curious. Resembled two stones united by a narrow band. *Vide* text.
19	July 15th	M.	8	,,	,,	,,	Stone very large; weight 7 drms. 38 grs.
20	Nov. 4th	M.	5	,,	,,	Death on 22nd day	Scarlatina 2 days after operation. Stone small; weight 30½ grs.
	1869.						
21	May 26th	F.	10	Vesico-vaginal	,,	Recovery	—
22	June 16th	M.	8	Lateral (?) lithotomy	,,	,,	Three calculi extracted.
23	Oct. 20th	M.	9	,,	,,	,,	—
24	Dec. 1st	M.	5	,,	,,	,,	—
	1870.						
25	July 16th	M.	6.6	,,	Mr. Marsh	Good	Had two attacks of hæmorrhage and had varicella.
26	Aug. 13th	M.	2.6	Lateral	Mr. Haward	,,	Had erysipelas, scarlet fever, and measles.
27	Dec. 8th	F.	3	Vesico-vaginal	Mr. Marsh	Relieved	Fistula remained, and she caught smallpox. Fistula was afterwards successfully closed.
	1871.						
28	Feb. 8th	M.	4.6	Lateral	Mr. Smith	Recovery	Hæmorrhage from wound on 2nd day.
29	April 19th	M.	11	,, (?)	Mr. Marsh	,,	Had hæmorrhage, and also an abscess in perineo

30	June 21st	M.	2.6	„ (?)	Mr. Smith	„	—
31	Sept. 16th	M.	4	„ (?)	„	„	Caught scarlet fever; albuminuria.
32	„ 23rd	M.	11	„ (?)	Mr. Haward	„	Second operation (first April 19th, 1871). Small oval stone removed. Wound from first operation had not closed. Had great pain in both hips and some slight peritonitis.
	1872.						
33	April 20th	M.	7	„ (?)	Mr. Smith	„	Got scarlet fever 2 days after coming into hospital. After operation a great deal of pus in urine and albuminuria as a consequence.
34	May 4th	M.	5	„ (?)	„	Good	—
35	Aug. 10th	M.	3	„ (?)	Mr. Marsh	Cured	—
36	Sept. 9th	F.	—	Lithotomy (?)	Mr. Smith	Successful lithotomy	Uric acid calculus coated with large quantity of phosphorus. There was difficulty in removing stone because of this soft crusting.
						Partial success with vesico-vaginal fistula	The incision was made in anterior wall of vagina, which was afterwards adjusted with five sutures.
37	Nov. 23rd	M.	8	Lateral (?)	„	Successful	Was under Mr. Smith in St. Bartholomew's Hospital 3 months ago. No stone could be detected at that time.
	1873.						
38	Feb. 2nd	M.	4.6	„ (?)	„	„	Symptoms had lasted only 3 weeks. On the Sunday, day of operation, they were so urgent as to require immediate operation. Small stone.
39	June 18th	M.	6	„	„	„	—
40	Oct. 17th	M.	3	Incision of urethra	Mr. Marsh	„	Calculus could easily be felt in its position. Mr. Marsh seized it with his fingers and cut down into urethra, whence it was easily got out.
	1874.						
41	March 5th	M.	8	Lateral (?)	Mr. Smith	Death	Had had the stone in the bladder about 7 years. Death from old abscesses in the kidney.
42	„ 25th	M.	4.6	„ (?)	„	Recovery	—
43	May 2nd	M.	3.9	„ (?)	Mr. Marsh	Successful	—
44	July 15th	M.	5	„ (?)	„	„	—
45	Oct. 31st	M.	2.6	„ (?)	„	Recovery	Stone very large; weighed 3 oz. 2 scr. 4 grs.
46	Dec. 2nd	F.	4.4	Vesico-vaginal	Mr. Smith	Successful	—

No.	Date.	Sex.	Age.	Nature of operation.	Operator.	Result.	Remarks.
	1875.						
47	April 14th	M.	5	Lateral (?)	Mr. Smith	Recovered	—
48	June 12th	M.	2	,, (?)	Mr. Marsh	Death	Child very feeble. Pleurisy; diarrhœa.
49	July 3rd	M.	2.6	,, (?)	,,	Recovery	Two calculi.
	1876.						
50	Jan. 8th	M.	3	,, (?)	,,	Successful	—
51	,, 5th	F.	5	Per urethram	Mr. Smith	,,	No incontinence after operation. Had been cut (laterally) for stone 13 months previously.
52	Feb. 23rd	M.	3	Lateral (?)	,,	Recovery	—
	1877.						
53	Feb. 28th	M.	9	,, (?)	Mr. Marsh	,,	—
54	March 10th	M.	2	,, (?)	,,	Died	Pyæmia.
55	July 7th	M.	2.6	,, (?)	Mr. Smith	Recovered	—
56	Oct. 17th	F.	2.9	Vaginal lithotomy	,,	Successful	Stone removed through roof of vagina. No incontinence of urine.
57	,, 30th	M.	11	Lateral (?)	Mr. Marsh	,,	—
	1878.						
58	April 17th	M.	9.6	,, (?)	Mr. Smith	Recovery	Oxalate of lime calculus.
59	June 19th	M.	4	,, (?)	,,	Successful	—
60	Oct. 6th	M.	5.5	,,	,,	,,	Stone, uric acid; weight 2 oz. 2 scr. 18 grs.
	1879.						
61	Feb. 8th	M.	8	,, (?)	,,	,,	Uric acid calculus; weight 1 oz. 4 grs. Orchitis 18th day. Stitches removed April 30th. Discharged May 1st.
62	April 2nd	F.	5.6	Vesico-vaginal	,,	,,	Not a trace of fistula. No incontinence of urine.
63	,, 16th	M.	6	Lateral	,,	,,	Uric acid calculus; weight 151 grs. Orchitis 16th day; lateral lithotomy. Mr. Smith held staff himself.
64	June 25th	M.	2.3	,,	,,	,,	Weight 108 grs.
65	Sept. 10th	M.	4	,, (?)	,,	Recovered	Oxalate of lime, 36 grs.

66	Oct. 4th	M.	11.9	„ (?)	„	„	Uric acid calculus, 140 grs. Two oval lithic acid stones.
67	„ 29th	M.	2.3	„	„	Successful	—
	1880.						
68	Sept. 1st	M.	3.8	„	„	„	—
	1881.						
69	Feb. 16th	M.	11	„	„	„	—
70	„ 16th	M.	6.2	Per urethram	Mr. Owen	„	—
71	March 19th	M.	6.11	Lateral	Mr. Marsh	„	Two stones were found at the operation weighing together 45 grs.
72	May 14th	M.	9.7	„	Mr. Smith	„	—
73	„ 18th	M.	2·3	„	Mr. Marsh	„	One large stone and one small.
74	July 13th	M.	3.3	„	Mr. Smith	„	—
75	Sept. 3rd	M.	1.8	„	Mr. Owen	„	Straight staff used.
76	Oct. 29th	M.	2.9	„	Mr. Marsh	„	—
	1883.						
77	Sept. 8th	M.	?	„	Mr. Owen	„	—
	1884.						
78	June 11th	M.	7	„ (?)	„	„	Three stones removed. Much trouble after operation from atony of bowel and accumulation of fæces.
	1885.						
79	Jan. 24th	M.	9.5	„	Mr. Morgan	„	Ten months previously had passed a small stone per urethram.
80	March 11th	M.	5	„	Mr. Owen	„	—
81	June 20th	F.	2.3	Lithotrity	Mr. Pitts	Successful, partially	None of the nucleus seems to have been crushed; only the outer laminæ of phosphates came away.
81A	„ 27th	F.	2.3	„	„	Successful	Second operation. The nucleus of stone was crushed; appeared to be lithic acid.
82	July 29th	M.	11	Supra-pubic	Mr. Morgan	„	Sept. 28th, went to Ascot. Small fistula still remains, which discharges slightly. Nov. 19th, sinus now healed.
83	Sept. 9th	M.	3.6	Lateral	Mr. Pitts	Recovery	Stone, about size of almond, removed.

No.	Date.	Sex.	Age.	Nature of operation.	Operator.	Result.	Remarks.
84	Sept. 19th	M.	3.10	Calculus in urethra	Mr. Owen	Recovery	The calculus could be felt in perineum. Mr. Owen cut down upon it in the middle line, and extracted a small stone about the size of a pea.
85	„ 30th	M.	3.6	Lateral (?)	„	„	Stone, about size of small eating chestnut, removed. Uric acid.
86	Nov. 21st	M.	2.6	Supra-pubic	Mr. Morgan	„	Size of stone 243 grs. Uric acid. Boy had diphtheritic paralysis in December. His condition for a time was very critical.
	1886.						
87	March	M.	8.2	Lateral lithotomy (?)	Mr. Marsh	Successful	Small stone, about size of small almond. Mar. 24th, N.B. with scarlet fever.
88	April 17th	F.	5.5	Lithotrity	Mr. Owen	„	Mr. Owen dilated the urethra, and crushed the stone with a lithotrite. The stone was supposed to be about the size of a Spanish olive.
89	Sept. 18th	M.	8	Supra-pubic	Mr. Marsh	Cure	Slight extravasation of urine after vomiting; much urine passed through wound. Wound healed and patient up in 13 days.
90	Oct. 21st	M.	10	Lateral	Mr. Owen	Successful	—
91	„ 23rd	M.	6.5	Supra-pubic	Mr. Morgan	„	'Lancet,' May 7th, 1887.
	1887.						
92	Jan. 19th	M.	5	Lateral	Mr. Owen	„	Considerable hæmorrhage after operation, and a little more on 8th day.
93	Feb. 12th	M.	4.3	Supra-pubic	Mr. Marsh	„	—
94	„ 16th	F.	5.9	Lithotrity	Mr. Owen	„	—
95	June 11th	M.	1.4	Supra-pubic	Mr. Morgan	„	Bladder was not stitched. Wound healed on 6th day.
96	July 12th	M.	3.3	Lithotrity	„	„	42½ grs. came away at the time of the operation; 1 gr. was passed 2 days afterwards.
97	Sept. 7th	M.	9	Supra-pubic	Mr. Pitts	Recovery	One calculus was found in mouth of ureter.
98	Oct. 26th	—	—	Lateral (?)	Mr. Owen	Successful	Stone about size of small hazel-nut.
99	Nov. 3rd	M.	2.8	Lithotrity	Mr. Morgan	„	Nov. 5th, walking about ward. No pain or any other trouble in micturition since operation.

100	Jan. 11th	M.	3.6	„	„	Cured	Calculus, uric acid, 54 gr. Some pain in micturition for 3 days after operation. A very little gravel passed during first 2 days. Got up on 5th day.
101	Feb. 11th	M.	5.5	Supra-pubic	Mr. Pitts	„	Bladder sewn completely up. Skin wound left open at lower part, with drainage tube inserted. Urine leaked through wound on 4th day; skin sutures removed, and wound opened in whole extent; boracic fomentations applied. Urine ceased to come through wound on Feb. 26th, and wound had healed by March 7th.
102	April 2nd	M.	5.6	„	Mr. Marsh	„	Bladder wound sutured with chloric carbolised gut; skin wound left open ½ in. at lower part for drainage tube; incision opened left rectus sheath; bladder united thoroughly. Some suppuration of superficial wound, probably from rectus sheath. Wound healed April 23rd.
103	„ 11th	M.	4.2	„	Mr. Morgan	Death April 14th, 1888	Boy suffering from diarrhœa at time of operation, which had not been reported by nurses. Bladder sewn up with gut; skin wound sewn up, with drain at lower end. Urine through wound on day of operation Boy collapsed, but temperature rising. P.M.—Peritonitis. Lithotrity previously tried March 28th, but urethra too small for lithotrite.
104	May 12th	M.	3.7	„	Mr. Marsh	Cured	Second stone not diagnosed before operation. Bladder opened in usual way and stones easily removed; bladder wound united with seven catgut sutures; skin wound united above with catgut. Left open below for drainage tube, which was removed after 24 hours. Urine leaked through wound on May 15th; ceased to come through wound on May 19th. Healed May 29th.
105	July 25th	M.	4	Lithotrity	Mr. Owen	„	—
106	„ 30th	M.	8.10	Supra-pubic	Mr. Morgan	„	Bladder wound left open. Urine first came through penis on 12th day.

No.	Date.	Sex.	Age.	Nature of operation.	Operator.	Result.	Remarks.
107	Sept. 10th	M.	4.5	Supra-pubic	Mr. Marsh	Cured	Small uric acid calculus. Some oozing of blood from wound during first 24 hours. Healed soundly by first intention. No escape of urine through wound.
108	Oct. 20th	M.	5.2	,,	Mr. Morgan	,,	Stone very large, broke in extraction. Wound in skin and bladder left open.
109	Nov. 7th	M.	9	,,	,,	Death Nov. 8th, 1888	Lithotrity begun, but stone too large for lithotrite. Removed by supra-pubic incision, which was left open, and fragments washed out. Bled during day, so that chloroform had to be given again in evening and two vessels tied. Vomited constantly next day. Died 8.45 p.m. P.M.—No very evident cause of death; septicæmia.
110	,, 28th	M.	3.2	Lateral	Mr. Owen	Cured	Lithotrite could not be introduced; urethra small. A stone was removed, but wound remained unclosed for some weeks. Patient had nasal diphtheria, and was in separate ward for 5 weeks. Sinus from bladder completely healed over.
	1889.						
111	July 31st	M.	5	Lithotrity	,,	,,	A small stone, the size of a small nut, was crushed and evacuated.
112	Aug. 3rd	M.	3.6	,,	Mr. Morgan	,,	The stone was about the size of a pea, and was crushed and evacuated.
113	Oct. 18th	M.	3.8	,,	,,	,,	A small stone, about the size of a pea, was crushed by the usual operation.
	1890.						
114	Jan. 22nd	M.	5	,,	Mr. Owen	Death	Bladder ruptured. Laparotomy and perineal cystotomy.

Note.—These statistics are copied verbatim from the operation-book of the hospital. A (?) is placed in those instances where the nature of the operation is not specified.

CLINICAL OBSERVATIONS

UPON

RESPIRATION DURING ANÆSTHESIA;

WITH

SPECIAL REFERENCE TO THE CAUSES OF EMBARRASSED AND OBSTRUCTED BREATHING.

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Introduction.

So much has lately been written concerning anæsthetics and their physiological effects, that I feel the greatest diffidence in bringing the following observations before this Society—a diffidence which is the greater when I reflect that the Royal Medical and Chirurgical Society has itself done much important work in this particular department of medicine. At the present moment, too, the Report of the Hyderabad Chloroform Commission is before us, and I am afraid it may appear almost presumptuous on my part to submit any facts or theories to the consideration of the Society so soon after the publication

of the important conclusions of that Commission. But the remarks I am about to make are solely the outcome of clinical as opposed to experimental work. It may be said that our store of knowledge concerning the effects of the anæsthetics, with which we are now acquainted, may be increased in two ways: firstly, by direct physiological experiment; and secondly, by clinical observation. With regard to the former mode of progress, I have unfortunately nothing to say. With regard to the latter, however, it seems to me that every one who is specially engaged in administering anæsthetics should endeavour, by making known the symptoms which he is in the habit of observing under particular conditions and circumstances, to contribute to the solution of the problems with which we are at the present time surrounded. The Hyderabad Commission has done a most important service in drawing attention to the necessity for carefully watching respiration during anæsthesia. I do not here propose to discuss the conclusions of the Commission; time itself will be the best judge of their correctness. That respiratory embarrassment or failure frequently precedes cardiac depression under chloroform there can be no doubt; but that such a sequence of events invariably occurs is still, I venture to think, an open question. Apart from this point, however, we must all admit the necessity of maintaining free respiration during anæsthesia; and it is with the hope that some light may be thrown upon the nature and causes of those modifications in respiration, which occur during anæsthesia in the human subject, that I venture to place the following observations on record. In order to avoid respiratory difficulties it is, in the first place, essential that the nature of those difficulties should be understood. I think it will be admitted that there are many conditions in the human subject capable of modifying respiration, which have no true counterpart in the lower animals. Some patients are said to take anæsthetics badly, whilst others are said to take them well. Generally speaking a patient is said to take an anæsthetic badly when the

respiration becomes embarrassed or impaired during the administration. It, therefore, seems to me a matter of the greatest interest and importance to attempt to discover some reasons for the numerous alterations and modifications in respiration which may be met with in actual practice. I am aware that such an attempt is, in the present state of our knowledge, unlikely to be attended by much success. Still, there can be no doubt that if we could discover the reasons of those phenomena, which we from time to time meet with, more especially in difficult and exceptional cases, we should be acquiring information, which might possibly be of future service in preventing those fatalities under anæsthetics, which we are all so anxious to avoid.

The respiration at any particular moment during anæsthesia for a surgical operation will be found to be influenced by one or more of the following considerations :

1. *The anæsthetic employed.*

2. *The method of its administration,* the most important factors in this connection being ; (*a*) *the rate of administration,* and (*b*) the degree of air-limitation permitted.

3. *The depth of anæsthesia,* for example, the act of deglutition, which cannot occur in profound narcosis, often modifies respiratory rhythm in the earlier and later stages of the administrations.

4. *The nature of the operation*—during the performance of certain operations, such, for example, as ligaturing hæmorrhoids, respiration is sometimes temporarily interfered with by reason of reflex spasm of the laryngeal and other muscles, a condition more especially likely to arise when the anæsthesia is only moderately deep.

5. The presence of any *pre-existing or accidental impediment within the air passages* to the free entry or exit of air ; these will be particularly considered later on (see § 1 and 2).

6. The presence of *any other condition capable of hindering free expansion of the lung,* such as extreme obesity, abdominal distension, pleural or pulmonary diseases,

senile changes in the framework of the chest, tight lacing, &c.

7. *The activity of the nervous mechanism of respiration.*

The above list very imperfectly represents all the circumstances which may influence respiration during anæsthesia. I have, for example, purposely omitted to refer to the effects upon the respiratory centre of impulses passing along the vagi or other nerves, feeling that any allusions to such points would at once destroy the object of the present paper, which is to consider the practical, rather than the physiological, side of the subject.

§ 1. *On the chief Morbid Conditions capable of prejudicially affecting Respiration during Anæsthesia.*

Any pre-existing inadequacy in, or impediment to, respiration should be carefully investigated before administering a general anæsthetic. Moderate or occasional dyspnœa prior to the administration is, for many reasons, liable to pass into considerable difficulty of breathing during anæsthesia, and there are undoubtedly certain cases in which no anæsthetic should be given. Morbid growths within the upper air-passages may increase in size by reason of greater vascularity, principally of venous origin. Such growths, moreover, may, if pedunculated, alter in position, and so give rise to sudden symptoms of an asphyxial character. Should nasal polypi or other formations be present to such an extent as to partially obstruct the nasal air-way, it will often be found that when the patient is deeply under the anæsthetic the nasal obstruction becomes complete from vascular turgescence of the growths, and oral breathing has to be maintained. I have observed a similar result in ordinary nasal catarrh. It is sometimes necessary to give an anæsthetic to a patient in whom considerable and possibly long-standing dyspnœa, depending upon some obstruction within the larynx or trachea, is present. Such cases require special notice. When extraordinary muscles of respiration have been

brought into play in the course of any disease, producing a narrowed air-way, the administration of an anæsthetic may, by throwing such muscles out of action, readily lead to arrested breathing. This event has been observed in cases of aneurysmal pressure upon the trachea, three fatalities having been recorded. In each of these respiration came to a final standstill after but a small quantity of the anæsthetic had been cautiously administered. Respiratory embarrassment from morbid or senile changes within the bronchi, lungs, pleuræ, or thoracic framework is not likely to eventuate in any serious conditions if due care be exercised. In order to maintain respiration satisfactorily under these difficulties three points must be borne in mind. The anæsthesia should never be very profound; a free supply of air should be allowed, and every means should be employed to maintain a good and efficient action of the heart. Those cases would appear to be the most unfavourable in which the respiratory embarrassment is of recent origin, and is, in part, dependent upon the thorax or abdomen (as the case may be) having become useless for respiratory purposes. The effects which impeded or inadequate respiration quickly produce upon the circulation, more especially if the latter be feeble, will be considered in § 4. Before quitting this branch of the subject, however, I would refer to a curious state of the respiratory mechanism which has been noticed in certain cases in which during anæsthesia breathing has come to a standstill, owing to the presence of some pre-existing morbid condition. There has been observed a peculiar rigidity and fixity of the thoracic parietes resisting all artificial attempts at expansion, even though no obstruction between the lungs and external air existed at the moment when such attempts were being made. Two cases of this kind are on record (see 'Brit. Med. Journ.,' March 15th, 1884, p. 508, and May 2nd, 1885, p. 887). At one of these reported by Mr. Holmes I happened to be present, and was able to watch the symptoms which occurred. In each case an enlarged thyroid gland pressed upon the

trachea; respiration became embarrassed under ether (probably from increase in size of the thyroid), and eventually ceased; tracheotomy was performed, and although by this operation an air-passage to the lungs was established, no efforts were successful in producing expansion of the chest, which appeared to be rigidly fixed in consequence of spasm of the expiratory muscles. In the case published by Mr. Holmes the heart's action continued for a considerable time whilst attempts were being made to expand the chest.

§ 2. *On the chief Accidental Obstructions and Impediments to Respiration during Anæsthesia.*

During anæsthesia the entry and exit of air to the lungs may become temporarily interfered with, either by reason of some alteration within the upper air-passages leading to narrowing or actual occlusion of the air-way, or by reason of tonic or clonic spasm affecting muscles normally engaged in maintaining efficient lung expansion. The latter condition, although of interest, is not of sufficient importance to need further remark, and I shall, therefore, only consider the former causes, as they are of much greater consequence.

Although it is true that accidental hindrances to respiration may, with a little management on the part of the administrator, be quickly corrected, cases are sometimes met with in which the respiratory embarrassment is of a more obstinate nature. In order that anæsthesia may be safely maintained, it is absolutely essential that a free air-way from the lungs to the external air shall be present throughout the administration, and profound stertor, or any other sound indicative of the commencement or establishment of obstruction, should be carefully watched for. It is often said in describing the symptoms of a particular case that the patient "stopped breathing," an expression which usually means that some impediment to the free passage of air suddenly gave evidence of its

occurrence. Putting out of the question the presence of adventitious bodies we may say that, when respiration becomes accidentally obstructed, such obstruction will be due either to—

1. An altered relation between the parts constituting the boundaries of the upper air-passages;
2. Vascular engorgement and swelling of those parts; or
3. To both of these conditions combined.

1. *Altered Relations between the Parts constituting the Boundaries of the Upper Air-way.*

Sometimes, more especially in the completely relaxed subject, certain parts may fall together, as when the lips of an edentulous patient meet, or the flaccid tongue and lower jaw fall backwards. On other occasions, more especially during the period immediately preceding deep anæsthesia, the obstruction is due to reflex spasm of muscles whose contraction leads to a diminution in the size of the air-way, such spasm being due, either to too strong an anæsthetic vapour or to operative procedures upon extremely sensitive parts. Respiration, which usually takes place wholly or partially through the nose during anæsthetic sleep, is often rendered difficult or impossible by allowing the head to be bent too much forwards. In this position the soft palate will meet the pharynx, and if the mouth be closed and the tongue applied to the teeth respiration will at once cease. Moreover, tilting forwards the head may have the same effect as depressing the lower jaw, that is, it may bring the base of the tongue and epiglottis into contact with the pharynx and so prevent either nasal or oral respiration. The stoppage of breathing, which sometimes occurs when the mouth is opened by a Mason's gag, is due to this cause. In the next place respiration is frequently interrupted and sometimes suspended for a brief period by the closure of the larynx during an act of deglutition. Deglutition is often performed in separate stages, and, thus it happens, that the

larynx may remain applied for a time to the epiglottis, rendering respiration impossible till descent of the larynx has taken place. The completion of the act of swallowing is most likely to be delayed for a considerable period, in other words, respiration is most likely to be suspended from this cause for a considerable period, just as the patient is entering upon, or emerging from, the state of deep anæsthesia. The apnœa thus induced is usually capable of correction either by pushing the lower jaw forwards from behind, by raising the chin, or (less conveniently) by the so-called complete extension of the head and neck. By the kindness of Dr. MacDonald and Mr. Harvey (at the Hospital for Diseases of the Throat) I was enabled to watch, in a patient anæsthetised and relaxed by ether, the readiness with which, by simply pushing the lower jaw forwards from behind, the epiglottis could be made to leave the larynx. Whilst examining the throat of an etherised patient I was also able to satisfy myself of the uselessness of forcible tongue traction in moving the epiglottis—a point which Sir Joseph Lister has, from observations upon his own throat insisted upon, and one which other observers, by experiments upon the cadaver, have corroborated. Pushing the lower jaw forwards from behind usually suffices to remove any obstruction due to the epiglottis. Sometimes, however, there co-exists with the half-performed act of deglutition much tonic spasm of the muscles of the neck, jaws, floor of the mouth, and other parts, rendering remedial measures difficult to apply, and under these circumstances neither pushing the lower jaw forwards nor extending the head and neck may be practicable. When breathing is thus arrested the mouth must be at once opened with some form of gag in order to admit air. The preliminary insertion of a small mouth gag, in those cases in which this difficulty is likely to arise, is strongly to be recommended. The so-called "complete extension of the head and neck" (which undoubtedly has the power of removing the epiglottis from the larynx in the relaxed cadaver) cannot be depended

upon for the relief of apnœa thus arising. There is usually considerable dissimilarity between the upper air-passages of an anæsthetised patient and those of the cadaver, as will be admitted when we take into account the absence of spasm and vascular engorgement in the latter. The part played by venous turgescence in causing stertor and in occluding the upper air-passages will be more particularly discussed presently.

2. *Vascular Engorgement and Swelling of Parts constituting the Boundaries of the Upper Air-passages.*

Stertor and respiratory embarrassment from this cause are more liable to arise in plethoric, florid, or stout subjects than in anæmic or fragile individuals. There is reason, indeed, for the belief that in the former patients temporary hindrance to respiration is often chiefly, if not exclusively, the outcome of vascular turgescence of the tongue, palate, pharynx, nasal cavities, and adjacent parts. The more plethoric the patient the more are stertor and temporary alteration in the rhythm of respiration liable to arise. Anæmic and cachectic persons, as well as those advanced in years, are not liable to exhibit respiratory difficulties from the cause referred to—a fact that is sometimes expressed by stating that such subjects "take chloroform well." The worst subjects for anæsthetics are bloated, flabby, and alcoholic patients, and the difficulties which arise during the administration to these patients are often due to the cause here indicated. This turgescence is doubtless of vascular origin, and is apparently chiefly venous in its nature. The tongue of a plethoric patient often becomes much swollen during anæsthesia, and by its increase in size obstructs the air-way. It has seemed to me, moreover, that the more air-limitation is practised in administering an anæsthetic to such subjects the greater the swelling and the greater the difficulties in keeping the air-way patent. The tongue of a short-necked, florid man under nitrous oxide, administered in the usual manner,

becomes greatly enlarged, but when the factor of oxygen-starvation is removed by administering 10 per cent. of oxygen with the nitrous oxide, the enlargement of the tongue will be found to be much less marked or even absent altogether. We can readily understand, with the facts which Dr. George Johnson has lately brought before our notice (see § 3), why air-limitation or deprivation during anæsthesia should lead to venous engorgement, and it seems but rational to infer that in full-blooded persons the accumulation of blood in the venous system would be more liable to cause noticeable turgescence of parts than in anæmic and thin individuals. I have on three occasions given anæsthetics to stout, full-blooded patients with glandular or other tumours at the root of the neck—tumours which undoubtedly interfered, by reason of pressure made upon them during operation for their removal, with the return of venous blood from the structures within the mouth. In each of these cases the tongue was greatly enlarged, and in one of them had to be kept half out of the mouth during the administration. The engorgement in question becomes of considerable importance when the air-way is previously encroached upon by morbid growths (see §1).

In this connection a few remarks must be made upon stertor, which, often affords timely warning of impending obstruction to the passage of air. Dr. Robert Bowles has brought many interesting facts concerning stertor before this Society. The soft palate may vibrate against the tongue during oral respiration, or against the pharynx during nasal respiration, and stertor may thus arise. Or the base of the tongue, more especially if the organ be congested and enlarged, may vibrate against the back of the pharynx and thus produce another variety. Then, as Sir Joseph Lister has shown, the arytæno-epiglottidean folds may vibrate and so cause another kind of stertor. The peculiar throat-sound of nitrous oxide narcosis will be more fully considered below. It seems probable that the venous turgescence, to which the tongue and other parts become liable under anæsthetics, is

an important factor in most forms of stertor. As a practical fact we know that the more plethoric the individual the deeper will be the stertor. When stertor is very profound the anæsthetic should be given cautiously, and the need for admitting air considered, whilst the lower jaw should be pressed well forwards from behind.

Before concluding this section I would refer to a curious condition which, so far as I am aware, has not been described, and which may be met with in connection with apnœa arising from accidentally obstructed breathing. It is well known that thoracic and abdominal movements will continue for a while after complete obstruction to the entry and exit of air has taken place. Now, if from any of the causes which have been mentioned, temporary obstruction at, or about, the superior aperture of the larynx should arise, the continuance of thoracic and abdominal movements will have the effect of increasing the difficulty by sucking the tongue or epiglottis more firmly into that position which originally led to this apnœa. It is hence very desirable that obstructed respiration should be immediately corrected, for the longer it persists the greater will be the difficulty in overcoming it.

§ 3. *On Certain Effects which may be produced by Diminished Oxygenation of the Blood (from Obstructed Respiration or other Causes) during Anæsthesia; with Remarks concerning the Respiratory Phenomena of Nitrous Oxide Narcosis.*

Though there are several objections to the view that general anæsthetics produce their effects solely by preventing or retarding the normal processes of oxidation within the central nervous system, there can be little doubt that the diminished oxygenation of the blood, which takes place in one way or another during the administration of these agents, often contributes very largely to the establishment of the anæsthesia with which we are familar in practice. The anæsthesia produced by ether is, for example, more

quietly, more quickly, and in the vast majority of cases more satisfactorily established when air-limitation is practised to a moderate degree than when the anæsthetic is administered with a free supply of air. This, indeed, explains the now almost universal plan of administering ether by what has been termed the close method. Numerous instances, moreover, might be given of the marked influence which a diminished supply of oxygen has in intensifying or prolonging the usual effects of anæsthetic agents. Although moderate air-limitation during anæsthesia is, generally speaking, attended by no untoward symptoms there are undoubtedly certain cases in which this factor should be carefully avoided. To these I shall presently refer. It will be well, before proceeding further, to mention the chief ways in which diminished oxygenation of the blood may arise during the use of general anæsthetics. It may result from :—

1. Intentional air-limitation, as when a bag inhaler is employed.

2. The anæsthetic vapour excluding air to a greater or less extent, as when ether is freely administered from a cone-inhaler.

3. Some special action of the anæsthetic upon the blood, as when by the administration of nitrous oxide the oxyhæmoglobin of the blood becomes reduced.

4. Some pre-existing condition mechanically preventing free access of air to the pulmonary capillaries, as pressure upon the trachea, pulmonary œdema, &c.

5. Some accidental embarrassment to respiration during the administration (see § 2) ; or—

6. Inadequate respiratory movements, as when the nervous mechanism of respiration becomes paralysed, or the bony framework of the chest from senile changes or morbid states of the lungs or pleuræ expands imperfectly.

In administering anæsthetics, one is frequently reminded that the action of the heart, more especially if that organ be functionally or organically deranged, is closely dependent upon the manner in which respiration is being per-

formed. Any marked interference with the respiratory functions—whether such interference be due to the presence of an obstruction, whether it arise from an absence of sufficient oxygen in the gas or vapour, which is being inhaled, or whether it be produced by the use of an inhaler which is not suitable to the case—will tend to depress an already enfeebled heart. Many instances bearing upon this point might be given. In illustration of the effects of temporarily obstructed breathing, I may mention that it is not an uncommon thing, when feeble old persons are under chloroform, for the slight interference with respiration incidental to the insertion of a mouth-gag, to be immediately followed by weakness or irregularity of the pulse, and for such symptoms to quickly vanish when the impeded breathing is relieved by pushing the lower jaw forwards. As an instance of the effects which may be produced upon a feebly acting heart by the inhalation of an anæsthetic free from, or nearly free from, oxygen, I may say that I have known symptoms of cardiac failure to manifest themselves during the administration of nitrous oxide to a young man, who was the subject of aortic regurgitation, symptoms, which were immediately dispelled by two or three compressions of the chest, the heart's action becoming restored immediately air was made to pass into the lungs. As an example of the deleterious effects of an unsuitable inhaler, I may direct attention to cases which have been recorded in which alarming or even fatal symptoms arose during the administration of ether to extremely asthenic subjects by means of Clover's inhaler. The air-limitation thus brought into play, coupled with the stress which would be thrown upon the respiratory mechanism by making the patient breathe through the narrow channels of this inhaler, might easily combine to seriously depress an already enfeebled circulation. Dr. George Johnson has devoted much attention to the effects which air deprivation produce upon the circulation, and his views receive considerable support from what we observe in administering anæsthetics. The

deprivation of air to any great extent leads, as he has shown, first to a constriction of the systemic arterioles, and consequent over-distension of the left side of the heart, and then to constriction of the pulmonary arterioles and over-distension of the right cavities. When the quantity of oxygen which the blood receives during the inhalation of an anæsthetic becomes much reduced, or when the respiratory movements are from any cause so feeble that they do not materially assist in the maintenance of the pulmonary circulation, we have evidence of the correctness of Dr. George Johnson's views in the general venous distension which is present, and which is more especially noticeable in the cervical region. When nitrous oxide unmixed with oxygen is, for example, pushed to its fullest extent, we find that the tongue becomes swollen, the veins considerably distended, and that venous blood freely issues from a cut surface; whereas if this gas be administered with 10 per cent. of oxygen, these phenomena, as well as others of similar significance (to which reference will be made below), are generally speaking absent. It may be said with regard to all anæsthetics at present employed, that, when from any cause the heart's action is weak or much embarrassed prior to the administration, the pulse will quickly show signs of failure should deficient oxygenation of the blood be allowed to occur to any considerable degree, an observation which applies with greater force to chloroform than to ether or nitrous oxide. The more vigorous the heart, the better, of course, will it be able to withstand the depressing influence of the deprivation of air. There is reason to believe that in many of the cases in which syncope has arisen during chloroform anæsthesia, the cardiac failure has in reality been the immediate result of embarrassed or impaired respiration. We are well aware that many fatalities with this anæsthetic have occurred during the period of excitement or during reflex respiratory embarrassment, *i. e.* whilst some degree of apnœa was present, and a considerable strain was being thrown upon the heart. Ether compares

very favorably with chloroform in this respect, for cardiac depression during excitement, and temporary apnœa under this anæsthetic are practically unknown.

We next come to the consideration of the respiratory phenomena of nitrous oxide narcosis. Respiration is deepened and quickened under this gas, the alteration in amplitude and rate of the respiratory movements being chiefly, if not entirely, due to the absence of free oxygen. After a variable though short period, the hurried but rhythmical movements undergo a peculiar and almost characteristic modification ; they become markedly arhythmical, and often a trifle slower. The alteration in rhythm is usually chiefly dependent upon repeated elevations of the larynx towards the epiglottis, by which the air-way becomes at irregular intervals momentarily occluded. In some cases, however, the rhythm of respiration becomes altered chiefly or wholly by reason of clonic contractions of thoracic and abdominal muscles, engaged in maintaining breathing. The peculiar throat-sound of nitrous oxide administration is due to the irregular elevations and depressions of the larynx towards the epiglottis. Other kinds of stertor (see last section) may, however, occur either independently of, or in association with, this characteristic throat-sound. The clonic contractions of the respiratory and extrinsic laryngeal muscles occur at or about the same time that clonic muscular phenomena in the extremities manifest themselves, and there can be little doubt that they are, like the latter, due to an absence of free oxygen. I have lately administered at the Dental Hospital of London for Dr. George Johnson pure nitrogen, as well as nitrogen with small percentages of oxygen, and the respiratory phenomena which have manifested themselves, as well as the general muscular movements, have so closely resembled those of nitrous oxide as to be indistinguishable from them. I have also conducted about 500 administrations of nitrous oxide with varying percentages of oxygen, and find that it is possible with 10 to 12 per cent. of oxygen to secure perfect anæsthesia without the occur-

rence of any asphyxial phenomena. With this percentage of oxygen, respiration never becomes jerky or laboured, the ordinary stertor of nitrous oxide is absent, there is no lividity of the features, and there are no clonic movements in the extremities. In many of the cases breathing becomes so calm as to be barely perceptible. I would strongly urge that when it is necessary to secure a brief period of anæsthesia in anæmic and feeble individuals, or in those suffering from any serious cardiac or pulmonary affection, nitrous oxide mixed with 10 or 12 per cent. of oxygen should be employed in preference to nitrous oxide alone.

§ 4. *On Failure of Respiration occurring independently of Obstruction or Other Impediment.*

It is not my intention to refer at any length to this form of respiratory failure. When respiration comes to a standstill, without any indication of impediment or obstruction, it is usually a matter of difficulty to decide what factors have principally contributed to the condition. The question is one which is, perhaps, more suited for physiological investigation than for clinical study. In its graver forms respiratory failure of this kind is fortunately rarely met with in practice, though by careful observation early indications of its approach may occasionally be detected. Speaking practically this form of cessation of breathing is in the vast majority of cases associated with a feeble or imperceptible pulse, and there are numerous reasons for regarding it, in many instances at all events, as the outcome of the latter condition, *i. e.* as due to cerebral anæmia. Thus profuse hæmorrhage during an operation may lead to arrested breathing, the respiration becoming re-established as cardiac action increases. It may be said, in fact, that syncope sometimes asserts itself by feeble or suspended respiration, a circumstance which has led many observers to overlook cardiac depression as a factor in respiratory failure in

the human subject. The undeniable effect of inversion in almost immediately restoring respiration which has become paralysed during chloroform inhalation is doubtless due to correction of cerebral anæmia. It is a significant fact that in a moderately healthy individual ether may be administered in very large doses, that is to say, till the pupils are widely dilated, all reflex action is abolished, and respiration is becoming feeble, shallow and irregular, without any such cardiac depression as that which occurs in the case of chloroform administered in similar toxic quantities. When respiration fails independently of obstruction from an overdose of ether it would seem that the failure is the direct effect of the anæsthetic upon the respiratory mechanism, the circulation remaining sufficiently good to negative the possibility of cerebral anæmia contributing to the suspension of breathing. The reason why resuscitation of the patient from an over-dose of ether is far easier than from an overdose of chloroform lies in the fact that in consequence of the intensely stimulating effects of the former anæsthetic, the heart's action is still maintained even though the respiratory mechanism has been paralysed by the anæsthetic.

In concluding this section let me add a few words concerning certain effects which may arise from the use of morphine in combination with general anæsthetics. Time will not permit me to enter at length into this matter. Respiration may readily become slow, feeble, or even arrested, from the combined effects of a very small quantity of morphine and general anæsthetics. Whenever the patient is even slightly under the influence of an opiate general anæsthesia must be induced with the greatest caution, and as a general rule it is best to administer the anæsthetic in very small quantities, and not to abolish reflex action.

Practical conclusions.

As the outcome of the foregoing observations the following practical conclusions may be deduced.

1. Any marked deprivation of oxygen (as by the incautious use of bag-inhalers) should be avoided (*a*) in patients with a very feeble circulation; (*b*) in persons who are the subjects of extreme obesity; and (*c*) in those whose respiratory apparatus has become seriously crippled by disease, in other words, when anæsthetising such patients no method of administration should be adopted by which the supply of free oxygen is greatly diminished.

2. Temporarily suspended respiration is far less likely to be attended by symptoms of cardiac depression under ether than under chloroform. The latter anæsthetic should, therefore, be more particularly avoided in those patients in whom excitement or reflex respiratory embarrassment is likely to be well marked; *i. e.* in those who are likely to require considerable quantities of the anæsthetic. There are, of course, many cases in which the operation could hardly be performed under ether (such for example as those of cleft palate and many operations on the mouth and nose), but even in these cases it is generally speaking better to *induce* anæsthesia with nitrous oxide and ether or the A.C.E. mixture, in other words, to tide over the initial stages with ether, and to *keep up* the anæsthesia with chloroform, than to administer the last-named anæsthetic from the beginning.

3. The initial difficulties in respiration which sometimes attend the administration of ether more especially in stout, muscular, or plethoric subjects, may be prevented by first gradually administering a small quantity of the A.C.E. mixture with a copious supply of air, and replacing this anæsthetic by ether from an Ormsby's inhaler, the change being effected when the respirations of the patient are becoming deep and uncontrolled, *i. e.* just before the stage in which muscular movements and other indications of

excitement sometimes arise. This method of producing anæsthesia has much to recommend it for general use, as it is simple to manage and the administration is not disagreeable to the patient.

4. In administering an anæsthetic the slightest impediment to respiration should be corrected as soon as possible in order to prevent an apnœa of a more abiding character from becoming established.

Appendix.

1. *References bearing upon* § 1.

'Brit. Med. Journ.,' March 29th, 1884, p. 645 (Bailey, on case of carcinoma of tonsil and on case of goître); 'Practitioner,' 1887, pp. 99—103 (Remarks on the use of anæsthetics in obstructive dyspnœa).

Of the fatalities which have occurred during anæsthesia in persons suffering from some pre-existing impediment to respiration the following may be referred to:—'Brit. Med. Journ,' March 18th, 1882, p. 380 (Tumour across trachea); 'Brit. Med. Journ.,' Feb. 24th, 1883, p. 351 (Aneurysm of aortic arch); 'Lancet,' August 8th, 1885, p. 245 (Innominate aneurysm); 'Brit. Med. Journ.,' Sept. 29th, 1888, p. 719 (Aortic aneurysm); 'Lancet,' Nov. 15th, 1884, p. 895, and 'Brit. Med. Journ.,' May 2nd, 1885, p. 887 (Bronchocele); 'Brit. Med. Journ.,' March 15th, 1884, p. 508 (Bronchocele); 'Lancet,' Sept. 1st, 1888, p. 442, and 'Brit. Med. Journ.,' Sept. 15th, 1888, p. 625 (Pharyngeal tumour causing dyspnœa); 'Brit. Med. Journ.,' May 19th, 1883, p. 978 (Fatal asphyxia probably due to growth of tongue); 'Lancet,' June 18th, 1881, p. 1014 (Diphtheria); 'Brit. Med. Journ.,' Feb. 23rd, 1884, p. 378 (Cancerous growth in gums and throat); 'Practitioner,' 1887, p. 101 (Innominate aneurysm); 'Brit. Med. Journ.,' July 13th, 1889, p. 88 (Stoppage of respiration after removal of tongue, case complicated by extensively adherent pleuræ); 'Lancet,'

June 25th, 1887, p. 1297, and 'Brit. Med. Journ.,' same date, p. 1397 (Pleural effusion); 'Brit. Med. Journ.,' Feb. 20th, 1886, p. 356 (Empyema); 'Brit. Med. Journ.,' Oct. 10th, 1868, p. 407 (Nitrous oxide in phthisis).

2. *References bearing upon* § 2.

'System of Surgery,' 2nd ed., 1870, vol. v, p. 492 (Lister on the effect of tongue traction, &c.); 'Trans. Roy. Med. and Chir. Soc.,' vol. xliii, 1860, p. 49 (Bowles on stertor); "Stertorous breathing in Apoplexy, and the management of the apoplectic state," by Dr. Bowles, published by the 'Brit. Med. Association,' 1881; 'Brit. Med. Journ.,' Jan. 12th, 1889, p. 65 (Bowles on raising the epiglottis); 'Trans. Roy. Med. and Chir. Soc.,' vol. xlvii, p. 403, cases 9, 14, and 17 (Recoveries from obstructed breathing under chloroform); 'Brit. Med. Journ.,' Feb. 14th, 1874, p. 201 (Clover on beneficial effect of raising the chin to remove epiglottis from larynx); 'Lancet,' Oct. 11th, 1879, p. 539 (Jacob on respiratory difficulties during etherisation); 'Lancet,' Oct. 27th, 1888 (Howard on raising the epiglottis); 'Brit. Med. Journ.,' Jan. 26th, 1889, p. 185 (On raising the epiglottis); 'Trans. Odont. Soc.' vol. iii, new series, p. 36 (Case of spasm of glottis under nitrous oxide); 'Trans. Odont. Soc.,' vol. iii, new series, p. 117 (Case quoted of stout plethoric man under nitrous oxide: tongue (probably from enlargement), causing obstruction to respiration); 'Lancet,' Feb. 16th, 1889 (Silk on "Anæsthetic Apnœa"); 'Brit. Journ. Dent. Science,' vol. xix, 1876, p. 229 (tonic spasm of respiratory and other muscles, probably of reflex origin and due to tooth extraction during imperfect anæsthesia, under nitrous oxide).

The following fatalities may be referred to as bearing upon the points discussed in § 2.—'Brit. Med. Journ.,' Jan. 8th, 1881, p. 62 (Struggling: laboured followed by obstructed breathing: cardiac failure); 'Lancet,' March

12th, 1881, p. 42, and 'Brit. Med. Journ.,' March 19th, 1881, p. 440 (similar case); 'Brit. Med. Journ.,' Feb. 24th, 1883, p. 350 (similar case); 'Lancet,' March 5th, 1881, p. 401 (similar case); 'Lancet,' July 29th, 1882, p. 153 (Tongue or epiglottis probably causing asphyxia); 'Brit. Med. Journ.,' Sept. 29th, 1888, p. 719 (Embarrassed respiration leading to fatal syncope); 'Lancet,' Sept. 3rd, 1881, p. 430 (similar case); 'Brit. Med. Journ.,' Nov. 22nd, 1884, p. 1027 (similar case); 'Trans. Roy. Med. and Chir. Soc.,' vol. xlvii pp. 377—396; see cases 17, 50, 79, 80, 83, 87, and 101; 'Trans. Odont. Soc.,' vol. ix, 1877, p. 182 (A death from nitrous oxide, probably due to the non-relief of obstructed respiration occurring during the administration).

3. *References bearing upon* § 3.

"An Essay on the Physiology of Asphyxia (Apnœa)," by George Johnson, M.D., F.R.S., 'Brit. Med. Journ.,' July 15th, 1882 (Lawson Tait on etherisation by close inhalers); 'Lancet,' April 27th, 1889, p. 832 ("On the Anæsthesia produced by the administration of mixtures of Nitrous Oxide and Oxygen").

The following fatalities may be referred to as bearing upon the points mentioned in § 3:—'Lancet,' Sept. 4th, 1880, p. 376 (Extreme asthenia: syncope during the arrest of breathing incidental to vomiting); 'Brit. Med. Journ.,' July 15th, 1882, p. 103 (Cardiac failure apparently due to asphyxia caused by the close method of etherisation in an asthenic subject); 'Brit. Med. Journ.,' Jan. 15th, 1881, p. 103 (Asthenia: cardiac failure during etherisation: two cases); 'Brit. Med. Journ.,' March 13th, 1886, p. 489 (quoted by Dr. Jacob: emaciated girl of ten years: syncope); 'Lancet,' Dec. 3rd, 1887, p. 1132, and the 'Lancet' article in the same number (Syncope apparently due to asphyxia produced by the administration of ether by close method to a cachectic patient with morbus

cordis); 'Journ. Brit. Dental Association,' Oct. 1880, p. 504 (Syncope under nitrous oxide); 'Journ. Brit. Dent. Association,' 1889, p. 689 (Syncope under nitrous oxide).

4. *References bearing upon* § 4.

"Anæsthetics, Ancient and Modern," by George Foy, F.R.C.S., 1889, pp. 105—109 (Cases bearing upon the effect of inversion in restoring respiration); 'Practitioner,' 1887, p. 103 (On morphine and general anæsthetics); 'Brit. Med. Journ.,' January 8th, 1881, p. 69 (Morphine and general anæsthetics); 'Brit. Med. Journ.,' June 14th, 1890, p. 1346 ("Remarks on the Report of the Second Hyderabad Chloroform Commission," by the Members of the British Medical Association Committee on Anæsthetics: see their remarks relative to cessation of respiration dependent upon feebleness of cardiac action).

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 33.)

NEPHROLITHOTOMY (FOLLOWING NEPHRECTOMY)

FOR TOTAL SUPPRESSION OF URINE LASTING FIVE DAYS.

COMPLETE RECOVERY AND GOOD HEALTH FIVE YEARS AFTER THE OPERATION.

BY

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The following case was mentioned by the editors of the medical journals at the time of the operation, in 1885, as a case of exceptional interest, but the details of the case have never been published, nor has the patient, or her kidney, or the stone which caused suppression, ever been exhibited. I have delayed publishing it because those to whom I mentioned it, whilst applauding the attempt to save a life on the extreme verge of dissolution threw the coldest doubt upon the patient's future; maintaining that even if she recovered from the immediate effects, her life must be a short and painful one; that the one remaining kidney having been opened and drained would rapidly

degenerate, or another stone would quickly form and bring about a final catastrophe.

I think now, after five years, sufficient time has elapsed to acquit me of any attempt to claim as complete an uncertain success. The patient is still living and enjoying to all appearances the best of health, and a freedom from pain, discomfort and hæmaturia, which, for seventeen years before her right kidney was removed, were almost constantly present.

In thus successfully operating for total suppression of urine after the removal of one kidney, it may, I think, be claimed that renal surgery has been pushed to its extreme limit of possibility, and the question of an operation on a single kidney has thereby been fully justified.

Much of the credit of the case is due to Mr. F. D. Atkins of Sutton, Surrey, who not only made the diagnosis of stone in the first instance, but by his promptitude in bringing the patient's dangerous symptoms to my notice, when suppression occurred, paved the way for her recovery.

In preparing to operate for the relief of this patient I carried out, what for years had been my intention, if any case of total suppression should come under my notice. It was not a sudden thought, but an operation the chances of which had been well considered; and I had publicly advocated its performance on more than one occasion. Thus in the 'British Medical Journal' of December 1st, 1883, I commented on a case published by Mr. Wilmot in the same journal of the previous week. This was a case of single kidney in which a stone caused symptoms of suppression for four days, and then was followed by suppuration and death apparently from septicæmia.

My comments on this case were as follows: "The case, published by Mr. T. Wilmot in the journal of November 24th, is eminently suggestive of operative interference in cases of suppression of urine. The necropsy revealed an absence of one kidney, and a small calculus blocking up the entrance to the ureter on the other side. A pre-

paration of a precisely similar nature is to be seen in the Museum of Guy's Hospital; and I recently heard of a case of suppression of urine where this diagnosis was made, and it was subsequently verified by the passage of a small calculus and the relief of the symptoms.

The symptoms in Mr. Wilmot's case were remarkably characteristic, for they commenced with a sudden sharp shooting pain in the left lumbar region, striking down to the glans penis and the testes, and stoppage of the flow of urine, with a constant desire to void it, accompanied with great bearing-down. When seen two days later, these symptoms continued, and, in addition, tenderness was discovered over the region of the kidney, subsequently a fulness was also noticed in this region. During four days, he is said to have passed only two ounces of urine, and then the flow recommenced; but meanwhile the damage had been done; rigors, vomitings, with a temperature of 104·6° had occurred; and the patient sank apparently in a septicæmic condition, seventeen days after the commencement of the attack.

It is interesting to note that this patient had had two previous attacks of suppression of urine, one lasting thirty-six hours, the other forty-eight. It becomes, then, a matter of some difficulty to determine when is the proper time to operate, seeing that in some cases the calculus may pass out, and in others recede so as to allow again the escape of urine. But it must not be lost sight of that if the symptoms disappear without the passage of a calculus, the patient is still left in a dangerous state, and his kidney is probably undergoing gradual dilatation and destruction. With a previous history of suppression unaccompanied by the passage of a stone, I think the surgeon would be justified in operating without delay, and in any case I should not be inclined to let the case pass over the second day without attempting to give him relief by an incision through the loin. When it is fully understood how simple a matter an antiseptic incision through the loin may be, medical men under whom these cases

may fall will have less hesitation in resorting to operation, and many lives will, I trust, be thus saved in the future."

Mr. Bennett May, who had also previously advocated surgical interference in such cases, wrote in support of my contention in reference to this particular case. His interesting paper, however, did not appear in the journal until March 8th, 1884. It ends by a comment on Mr. Wilmot's final remark, "The usual treatment was followed in this case," and Mr. May says:—"Undoubtedly such was the fact, and, let me add, it was followed by the same result —death. Whether the proposed departure from the usual treatment will ever lead to any other result, time and experience must determine." That time is now passed, and the experience which was looked forward to has been acquired, through the patient whose case will be described in detail this evening.

F. F—, æt. 37, was first admitted into Guy's Hospital on June 22nd, 1885. Her father had suffered from gout and died from congestion of the lungs. Her mother, two brothers, and one sister died from consumption.

For seventeen years she had suffered from hæmaturia at intervals, and for nine or ten years this had been accompanied with pain in the right side, and for seven years a tumour, diagnosed as a floating kidney, had been felt in the right side. On July 14th the right kidney was removed by a crucial lumbar incision. It was a mere shell containing masses of stone and weighing twenty-one ounces. The wound healed completely, and she left the hospital convalescent on August 10th, just within a month of the operation.

All went on well for three months. She had returned to her household duties, and I heard from time to time that she was free from pain and hæmaturia, and much satisfied with the result of the operation.

On Sunday morning, October 24th, 1885, she was suddenly seized, between 7 and 8 o'clock, with most violent and agonising pain in her back and left loin. The

pain passed through the loin to the front of the abdomen and groin. About 8 o'clock she passed a little urine, but from that time all secretion stopped. Vomiting commenced about half-past 8 on the same morning, and was continued at intervals and whenever anything was taken. Mr. Atkins was called to see her, and found the bladder empty. He prescribed medicines to stop sickness and diuretics, but everything was returned. Vomiting and anuria continued throughout Sunday, Monday, and Tuesday.

On Tuesday, October 26th, Mr. Atkins came to London to tell me that my case of nephrectomy was dying from suppression of urine due to some obstruction in the other kidney. I went to Sutton with him to see the case, and found, though the vomiting and anuria had caused great depression, she still had a good pulse and was in a condition to bear an operation. The bladder was quite empty. It was impossible to operate with any chance of success in her cottage, a long distance from her medical man, I accordingly tried to persuade the husband to bring her to London. He promised, if she got through the night, to bring her to Guy's Hospital on the following morning. She was admitted into the hospital on Wednesday, October 27th, in a very collapsed condition. The vomiting and anuria had continued since 8.30 a.m. on Sunday, but she still had a fair pulse. A catheter was passed, and the bladder still found quite empty. The pain in the loin and back was easier since yesterday morning but had not disappeared. She also complained of severe headache and giddiness, but said this was better than it had been. On the whole, we could not attribute any increase of symptoms to the journey of twenty miles to London.

It was my intention to cut down at once on the remaining kidney, but my colleagues, and more especially the late Dr. Moxon, advised me very strongly to give yet a trial to strong diuretics to try and wash the stone down. In deference to their opinion I resolved to wait till the following day, or until the patient was evidently dying. A tur-

pentine enema was given by the bowel, and a strong diuretic mixture containing acetate of potash by the mouth. Both were immediately returned and the vomiting continued all night.

On Thursday, October 29th, she still had passed no urine and was much worse. A tumour, evidently the hypertrophied left kidney, could be felt in the abdomen, and when she lay on the right side it fell forwards towards the middle line. It did not appear to have increased in size since Tuesday when it was first noticed. Towards the middle of the day she began to get drowsy, and it was difficult to rouse her to obtain answers to questions. Her pulse, too, was weaker, her temperature was 99°. It was clear that if anything was to be attempted it must be done at once.

The operation.—At 2.45 p.m. ether was administered by the House Surgeon (which the patient took very well), and an incision five inches in length was made about three quarters of an inch below the last rib and parallel with it. The external oblique, and latissimus dorsi, the internal oblique, and the tendon of the transversalis were divided in succession. Several small vessels were twisted and one ligatured with catgut. When the fascia transversalis had been divided, a large amount of perinephritic fat came into view, a good deal of which was cut away in order to expose the kidney more clearly. The kidney was then examined from behind with the finger. At first, no stone could be felt, but after a time at the lower part of the pelvis something rounded was felt, which slipped immediately away from under the finger and was lost in a calix. Being convinced that this was a stone movable in the pelvis, the wound was now enlarged and made crucial, whilst the kidney was pressed back into the wound. After some trouble the stone was again displaced into the pelvis, and being held there by the first and second fingers of the left hand, the pelvis was divided over it. In doing this, however, the stone again slipped away into its calix bed and was lost. The aperture in

the pelvis was then enlarged so as to admit the left forefinger, and a pair of curved forceps being inserted and guided by the finger the stone was seized and extracted. The pelvis was not found to be of any great size, but the kidney was large, and its tissue apparently healthy. Urine began to drip away as soon as the pelvis was incised.

The stone was rather more than three quarters of an inch in length, and in diameter, from three eighths to five eighths, and one inch and a quarter in its greatest circumference. From its appearance it will be seen it was exactly of the shape to act as a ball-valve to the top of the ureter.

A drainage-tube of large size was placed beneath the wound in the kidney and brought out at the lower part of the wound, and the remainder of the wound was closed with thirteen wire sutures. Before the dressings were applied, urine mixed with blood dropped freely from the drainage-tube. The wound was covered with gauze and iodoform, and a large pad of absorbent wool was applied outside. The operation lasted one hour and a quarter, and the patient recovered well from the effects of the anæsthetic.

The pads were changed at six o'clock the same evening owing to the escape of fluid from the wound. She was sick once at eight o'clock in the evening after the operation, but from that time no sickness occurred.

October 30th.—The patient passed a fair night, sleeping comfortably. The pads were changed five times during the night owing to the large escape of urine from the drainage-tube. No urine passed into the bladder. The wound was dressed and looked well. She has taken two ounces of milk and ice during the night. Temp. 99°.

31st.—The patient did not pass quite so good a night but feels no worse. The pads have to be constantly changed owing to the escape of urine. She takes milk, ice, and soda water without feeling sick. Temperature normal.

November 1st.—Still no water from the bladder, but the urine flows freely from the tube so that the pads have constantly to be changed. Wound looks well; ordered brandy ℥ij.

2nd.—Patient looks better, but complains of dry mouth and a feeling of dizziness. Temperature normal. No urine by the bladder; ordered chicken broth.

3rd.—Three of the stitches were taken out and the wound found to be healing well. She takes beef tea and jelly. All the urine still comes by the tube.

4th.—Patient had a good night's rest and feels better. Temperature normal. Wound dressed and more stitches removed. No urine passed from the bladder. She ate some boiled sole. Enema saponis was administered.

5th.—Passed a fair night, but complains of pain in the wound. After having slept for a time she gets into a cold perspiration and feels giddy. No urine by the bladder.

7th.—The remaining stitches were taken out, wound healthy. Temp. 99·2° last night. Still no urine by the bladder.

10th.—To-day for the first time since the operation the patient passed, *per urethram*, an ounce and a half of urine. It caused sharp cutting pain in passing, and contained a large amount of sediment. There was a large quantity of albumen, pus, epithelial-cells, and a trace of blood. The temperature last night was 99·4°.

13th.—The large tube was removed and replaced by a smaller one. She passed about two ounces of urine by the urethra.

15th.—Patient passed about ten ounces of urine by urethra this morning, she feels much better and looks brighter. The wound is granulating up well.

17th.—The patient passed in the last twenty-four hours twenty-nine ounces of urine, sp. gr. 1015, slightly alkaline in reaction, containing a trace of albumen, no blood or sugar, but phosphates and a small quantity of pus and epithelium.

18th.—Patient passed thirty-six ounces of urine in the last twenty-four hours of sp. gr. 1020, alkaline with a trace of albumen and a little sediment of phosphates, pus, and albumen. All the urine appears now to come by the natural channel. There is very little discharge from the loin, the dressings only requiring to be changed occasionally.

19th.—The patient passed forty-two ounces of urine to-day by the urethra. No urine now comes by the tube only a little discharge. The tube was shortened one and a quarter of an inch. The wound, through which the tube passes, is granulating up measuring two and a half inches by half an inch. The remainder has healed.

25th.—The patient has been making good progress. She has passed from thirty-two ounces to forty ounces of urine in the twenty-four hours since the last report. The tube has been gradually shortened and was to-day removed. There is very little discharge from the wound. Temp. 99° in the evening, 98·6° in morning.

From this time onward there was little to note. The trace of albumen and pus gradually disappeared from the urine. Quantity passed varied from thirty ounces to forty-four ounces.

December 20th.—The superficial wound was dressed with zinc ointment.

24th.—She sat up for the first time.

In January, 1886, the wound was completely healed, but she was kept in the hospital until February 14th, 1886, chiefly for the sake of observation, and because the weather was cold.

Remarks.—It will be noted that the symptoms commenced early on Sunday morning, and that it was not until the afternoon of Thursday, the fifth day, that the operation was undertaken. From a study of cases of obstructive suppression, previously reported, I was aware that the most serious symptoms seldom set in before six or eight days from the commencement of the obstruction, and this knowledge caused me to yield to the advice of

my medical colleagues to wait, even after the patient was admitted into the hospital, before attempting an operation which appeared to them must of necessity prove fatal. The case meanwhile was watched by a special nurse who had instructions to send for me if any convulsion, coma, or serious depression, showed itself. I waited until it became difficult to rouse the patient to answer questions, and until it was evident to all that her condition was getting desperate.

It will be observed that, as in obstruction of the intestine, the symptoms which were most urgent at the commencement tended to lessen as the case went on. The agonizing pain in the loin grew less, and might have led to the deceptive thought that the calculus had travelled on down the ureter. The vomiting also became less urgent, and the restlessness and sleeplessness gave way to a general sense of indifference, and the severe headache and giddiness almost disappeared. All this quiescence of symptoms pointed merely to the wearing out of the vital powers, and indicated no improvement in the patient's condition, whilst complete anuria remained. The size of the stone removed showed how hopeless it was to think that it could ever have passed down a ureter.

It was pretty clear that the stone had been a long time forming, but resting somewhere in the fleshy part of the kidney had given rise to no symptoms, until it dropped out of its bed and fell into the pelvis. This agrees with my general experience of renal calculus, which is that the fleshy substance of the kidney is comparatively insensitive, so that a fixed stone there gives rise to little symptom, but a stone, movable in the pelvis of the kidney and impinging on the outlet, causes the greatest agony.

Its mobility gave rise to a difficulty in detecting it, when I had exposed the kidney ; for the moment I touched it, it slipped away into its nest, where it could no longer be felt. When I again brought it into the pelvis by manipulating the cortex, I held it between the tips of two fingers of my left hand whilst incising the membrane over

it; but so easily did it move that before this was completed it had again disappeared. It was eventually turned out by the finger introduced through the pelvis, and removed by curved forceps.

It was interesting to note how rapidly relief to symptoms followed the opening of the kidney, and the free exit thus obtained for the urine.

The patient was sick once only after the operation, at eight o'clock the same evening. After this she fell into a refreshing sleep. From that time headache was no longer complained of, but a feeling of giddiness remained for several days.

The wound, in spite of the escape of urine, ran an antiseptic course, the temperature never reaching 100°, except once, six weeks after the operation, when she had a chill and her temperature rose for three days as in an ordinary cold.

It was not until twelve days after the operation that any urine was passed by the urethra, and I began to dread further obstruction lower down and a permanent urinary fistula in the loin. The urine first passed contained a thick sediment of pus, epithelium, phosphates, and some blood. It also caused great pain during micturition, at first only one ounce and a half passed through the bladder in twenty-four hours, but the quantity naturally voided rapidly increased after the sixteenth day, when the large drainage-tube in her loin was replaced by a smaller one. After the nineteenth day all the urine passed naturally, and none whatever was afterwards detected in the dressings. The wounds took about ten weeks to heal completely, and since that time there has been no sinus or any inconvenience from the operations on the two loins.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 42.)

DESCRIPTION OF PLATE II.

Nephrolithotomy following Nephrectomy for Total Suppression of Urine lasting Five Days (Mr. R. Clement Lucas).

A. Vertical section of the right kidney removed, choked with calculi, weighing twenty-one ounces. Reduced in size.

B. Conical calculus removed by operation, which blocked the orifice of the left kidney, and caused total suppression of urine. Actual size.

B
A

A CASE

IN WHICH

LARGE CALCULI WERE REMOVED FROM BOTH KIDNEYS,

FATAL HÆMORRHAGE FOLLOWING THE SECOND OPERATION.

BY

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Received December 12th, 1890—Read January 13th, 1891.

The following case, if described in full, would be a very long one; I propose, however, merely to report in detail those special facts which make it, as I think, worthy of the attention of the Society.

In November, 1888, I first saw, in consultation with Dr. Ralfe and Mr. Willans, a gentleman who was suffering from symptoms of renal calculus. He was a spare man, thirty-seven years old, 5 feet 8 inches high, and weighing 8 stone 7 pounds, this being somewhat less than his normal weight. He had led an active, indeed a somewhat athletic life, and had only complained of symptoms referable to the kidney for two and a half years, during

which time he had occasionally passed a good deal of blood, and, in the year 1887, a small stone. There was lumbar pain, principally upon the left side, but he felt sometimes what he described as a faint echo of this pain upon the right side. Both kidneys could be felt, but neither was apparently much enlarged. The urine was sufficient in quantity, but had a specific gravity of only 1010, and contained one-twelfth or more, by measurement, of thick ropy mucus and pus.

The diagnosis made, was—left renal calculus and left pyelitis; and very probably a calculus, if not pyelitis, on the opposite side.

On November 27th, the left kidney was exposed by the usual lumbar incision, and an enormous mass of stone, weighing 832 grains (moist), (732 grains dry), was removed from the pelvis and dilated calyces without material difficulty. The stones consisted of uric acid, coated with phosphates, and had a strong ammoniacal smell. The hæmorrhage was insignificant, although the substance of the kidney was, of necessity, very freely torn and divided. The shock of the operation was, though considerable, by no means alarming.

The points of interest during the healing of the wound are these:—

First. Though the urine in the dressings had a pungently ammoniacal smell, the discharges from the wound never became putrid, and the wound did not inflame.

Secondly. There was a copious erythematous rash—urticarial in character—which started from the wound, and then appeared all over the body, giving rise to most intolerable itching.

Thirdly. The urine passed from the bladder never became free from pus and mucus, and remained of low specific gravity. This was taken as a possible indication of the existence of mischief in the opposite kidney.

Fourthly. The wound healed with remarkable rapidity, being completely closed in about three weeks.

But this closure was not permanent. During the next

year he had a series of attacks marked by high temperature, drowsiness, sickness, and diminished secretion of urine, evidently depending upon the accumulation of pus and urine in the left kidney. They were sometimes relieved after the discharge of a plug of mucus by the urethra, sometimes by the opening of the lumbar wound. Once a stone was passed weighing ten grains by the urethra, and more than once from the wound, and ultimately it proved to be most convenient for him to wear an india-rubber plug in the sinus to keep it from closing, by means of which he could keep himself dry, and by the removal of which, when accumulation occurred, its unpleasant effects could be avoided.

In the summer of 1889, the soakage from the wound became constant and considerable, and I, therefore, assumed that the ureter was plugged, most probably by a stone, and on September 12th, I opened up the wound and explored the pelvis of the kidney, and the ureter as far as the brim of the pelvis. The whole ureter was very much thickened, and, at one point, was so hard that I fancied it must contain a stone, so I drew this part forward with my finger and a blunt hook, and made a small vertical incision into it; but it was clear that the hardness was due to the products of chronic inflammation and not to the presence of a calculus.

For some unexplained reason, at the moment of this exploration, rather severe hæmorrhage set in from the kidney which, at the time, was not being in any way interfered with. I could not see or secure the bleeding point, so it seemed best to plug the pelvis of the kidney, and I was obliged to desist from any further investigation of the ureter.

I cannot say why, but it is a fact that, after this operation, the ureter became again pervious, and the urine contained much less mucus and pus. Possibly the contact of the plugs for about a week may have had some beneficial effect upon the mucous membrane of the pelvis of the kidney. The patient returned to his work, wear-

ing only the india-rubber plug, which he removed from time to time to allow the urine to escape, and he improved very much in health and strength. But he was now able to devote his attention more to the other kidney, and he became convinced—and convinced me, too—that there was a stone on the right side also. I explained to him the risks of operating on a kidney when the other is known to be more or less disabled, and the possibility of the formation of a second permanent fistula, but I advised him to submit to it, and he, indeed, was anxious for the operation to be done.

On November 19th, 1890, the right kidney was exposed by a lumbar incision, and was at once felt to be full of stone. No hæmorrhage resulted from the incision of the kidney, but the removal of the stones (which weighed 480 grains dry), and the consequent free laceration of the renal tissue caused very smart venous bleeding, which, however, came from no obvious point and was always easily controlled by pressure.

At the end of the operation the pulse was very poor—so poor, indeed, that I was advised by the anæsthetist not to persist in my attempts at the removal of the last minute fragment of stone—but the bleeding had stopped, almost if not completely. I thought, however, with the experience of the last operation in my mind, that it would be wise to plug the pelvis, so I inserted rather firmly, round a large flanged drainage-tube, some strips of the cyanide gauze, and applied a firm bandage over a very thick dressing ; and when I left the house, half an hour after the completion of the operation, I did so with very little anxiety, for the pulse was slow and much stronger, and the general condition good.

When he awoke from the ether, he complained of intense pain, and begged to have the bandages released. This, of course, was not done. He remained in the same state for about an hour, when he suddenly collapsed, and at the same time, a considerable soakage of blood appeared through the dressings. I cannot, therefore,

doubt that hæmorrhage was the cause of the fatal result. It was not possible to make a post-mortem examination.

It is needless to say that this is a case, the record of which one would gladly conceal in his note-book and banish from his mind; but I think it should be published for the instruction of others. For, whilst the danger of wounding a large vessel in the kidney either by incision or laceration cannot be ignored, the bleeding, which is often free, so commonly stops of itself (as it does after incision into other vascular organs), that we are, perhaps, predisposed to underestimate its importance; but of a considerable number of cases (between twenty and thirty) in which I have incised the kidney, this is the first in which serious trouble has arisen from hæmorrhage, and the only one that has died as the immediate result of the operation. I gather from reading and inquiry that it is a very rare accident after nephrotomy.

In looking back at the case, I do not see that there were any indications to make me act differently at any particular stage of the operation; but, no doubt, it would have been wiser to have plugged the kidney more forcibly and without the use of a drainage-tube, if, indeed, it was wise to plug it at all. I do not believe that it would be possible to catch up and tie a large vessel in the kidney even if the organ were very widely opened and dragged up forcibly into the wound.

Perhaps it is the duty of the surgeon to wait for a longer time with his patient under such circumstances, or at all events to leave an assistant who is prepared to remove the dressings on the slightest suspicion of hæmorrhage; and, if necessary, to take out and re-introduce the plugs. It is easy to be wise after the event, and I can only hope that others will profit by my experience.

Postscript (June, 1891).—A very valuable suggestion was made by Mr. Arbuthnot Lane in the discussion that followed the reading of this paper, which I have myself since then put in practice, and can thus bear personal

testimony to its utility. He spoke of a case in which hæmorrhage from the cut surface of the kidney had been controlled by the pressure of a ligature, passed through the substance of the organ from front to back so as to close the particular part of the wound containing the vessel. I have, however, found even this to fail in the very congested kidney of a patient with complete suppression, caused by blockage of the ureter of a solitary kidney. The gland substance in this case was so brittle that it was impossible to exert sufficient force to tighten the ligature without making it cut its way out. In this case, however, the hæmorrhage before long stopped spontaneously. I cannot doubt that Mr. Lane's suggestion is a very good one.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 42.)

THE THERAPEUTICAL VALUE OF VENESECTION;

ITS INDICATIONS AND ITS LIMITS.

BY

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Received October 21st, 1890—Read January 27th, 1891.

The following remarks are based upon forty-nine cases in which I have ordered bleeding from the arm and have had opportunity of seeing its subsequent effect.

There is scarcely another instance of a remedy which was once so universally practised throughout the civilised world having fallen into such general disuse as venesection. It has often been said that our practice follows fashion rather than scientific indications, and it must be admitted that this reproach applies in some measure to the indiscriminate practice of blood-letting in the days of Abercrombie, Graves, and Latham, and to the equally indiscriminate abandonment of the treatment which followed. It is now generally believed that the routine employment of venesection in the treatment of inflammation was a mistake, that it does not possess the power attributed to it, and that it brings with it evils of its own. But if we regard its use in a more sceptical spirit than our forefathers and with the advantage of better physiological knowledge, we

may find a permanent place for the practice, secure from the changes of therapeutic fashion.

Bleeding from the arm is certainly more often ordered by my colleagues at Guy's Hospital as well as by myself at the present time than when we were students; and I have reason to believe that the same is true of hospital practice in London generally. But if there be such a revival of what was once an almost forgotten remedy, I am not aware of any record of the facts, and have therefore thought it might be of interest to record the experience of an individual.

Of the cases on which this paper is founded, thirteen were bronchitis, in most cases an acute attack supervening on emphysema, in several complicated by more or less of lobular pneumonia, and in one by acute miliary tuberculosis. Four were cases of acute lobar pneumonia, seven of valvular disease of the heart, one of primary cardiac hypertrophy, two of acute pericarditis with effusion, and two of adherent pericardium.

Eleven were cases of Bright's disease, two acute and the rest chronic. Six were aneurysm of the aorta, five of the arch and one of the abdominal trunk. Two were cases of apoplexy from cerebral hæmorrhage, and one was a case of epilepsy.

The ages of the patients varied from eight, thirteen, and fifteen up to seventy.

The result was in thirty-one cases marked relief; but only in twelve of these was there recovery, in five permanent, and in the other seven sufficient for the patient to leave the hospital. In nineteen cases the relief was temporary, but in several of these it lasted long enough for the urgent peril to be past, and for subsequent death to be fairly attributed to other causes. In eighteen there was only slight or no perceptible benefit.

In four of the above cases the bleeding was repeated on one or more occasions, and always with benefit.

The number of cases is far too small for statistical

treatment. Indeed, we are never justified in using percentages for numbers under 100. Moreover, the forms of disease were so different, and the age, the habits, and the condition of other organs than those primarily diseased varied so greatly, that no useful conclusion can be drawn from a mere enumeration of the results.

Venesection is not a remedy for the effects of any one pathological lesion. It is a *physiological* remedy, applicable to certain clinical conditions which may be the result of various causes, and is therefore comparable to purging or vomiting, not to mercury or quinine. The conditions in which blood-letting is indicated appear to be, speaking generally : 1st, *cyanosis* with dilatation of the right side of the heart; 2ndly, the pain of *aneurysm;* and 3rdly, eclampsia and other symptoms of *uræmia.*

Taking the first group of cases, classified in this way, no benefit was obtained in ten of them, while in fourteen the benefit was so striking and immediate that there can be little doubt the patient was rescued by venesection from immediate death. Very few, however, of these cases of bleeding in cyanosis were followed by ultimate recovery—only six out of the twenty-nine.

In the second group of cases, as well as in several other instances of aneurysm of which I have not notes, the relief was almost always very decided, the exceptions being chiefly where for some cause it was impossible to obtain so much as an ounce of blood.

In the third group of cases there was relief from the convulsions, or other uræmic symptoms which led to the employment of the remedy in nine cases, and in four of these recovery ensued.

I would submit to the Society the following indications of the value and limits of venesection, as justified at the present time both by physiological probability and by the test of clinical experience :

(1) General bleeding is useless or injurious as a remedy

for the condition of *fever;* it is counter-indicated in enteric and other specific fevers; it is injurious in acute rheumatism, and useless for hyper-pyrexia.

When the pulse is rapid, full, and as the old physicians expressed it "bounding," bleeding is in most cases injurious. Its effect is to lower the blood-tension and quicken the pulse, and, other things being equal, is indicated by a slow rather than by a frequent cardiac action.

(2) Bleeding is unnecessary and probably injurious in cases of lobar *pneumonia* or acute inflammation of the pleura, pericardium or peritoneum, of the pia mater or kidneys. Its effect on the inflammatory process is little if any, and it probably may lead to relapse or prolonged convalescence. When admissible in these cases, it is not as an antiphlogistic, but to relieve some complication such as pain or dyspnœa.

(3) Venesection is of very doubtful benefit in cases of *hæmorrhage*. By diminishing blood-pressure and the force of the cardiac beats it would theoretically favour cessation of bleeding and the formation of a clot. But this is just what the effect of the hæmorrhage itself would be. A very large hæmorrhage, as from a ruptured aneurysm or an artery opened by a gastric ulcer, probably proves fatal by syncope or by suffocating the patient, before there is time for treatment. Smaller hæmorrhages usually cease of themselves. Moreover the popular estimation of the danger of losing blood is much exaggerated. It is extremely rare for epistaxis, hæmoptysis, hæmatemesis, hæmaturia, or intestinal hæmorrhage to prove immediately fatal and in many cases, for instance, of phthisis we find the symptoms relieved after moderate hæmoptysis.

In the most formidable cases of hæmorrhage, those due, not to a local lesion but to such conditions as purpura, leuchæmia, and idiopathic anæmia of Addison, there is no reason to suppose that bleeding would do anything but add to the patient's danger.

(4) There is, however, one kind of hæmorrhage which is dangerous, not from the loss of blood, but from the local

mischief it produces—hæmorrhage within the cranium. In cases of *apoplexy* venesection was for many years regarded as the only effectual treatment, and remarkable instances are recorded of its good effects. Probably many of these were not due to cerebral hæmorrhage, but were epileptiform or uræmic. This criticism, however, can only apply to a certain proportion of the cases related, for instance, by so competent a pathologist as Abercrombie. The chief practical objection to the treatment is that the remedy must almost always come too late. Probably, however, venesection may often be useful in the condition of insensibility with cyanosis and stertorous breathing which frequently follows an attack of apoplexy—not by checking the cerebral effusion, but by relieving the distended right side of the heart which results from the embarrassed breathing.

(5) Bleeding in the most severe forms of *epilepsy* where convulsions are long continued or the patient passes into the status epilepticus with impeded respiration, and blueness of the surface is a plan of treatment which many years ago I saw practised with great success by Dr. Wilks. It has every probability to recommend it and is supported by experience.

(6) A similar condition in which bleeding is of undoubted benefit is that of eclampsia from *uræmia*, including puerperal convulsions and those of advanced Bright's disease. In these cases the left ventricle is hypertrophied, the resistance in the systemic capillaries is increased, and the pulse both to the finger and by a sphygmographic tracing shows high tension, with diminished systolic expansion and diminished diastolic relaxation. Under these conditions there is frequently severe headache and vomiting, with epistaxis, retinal hæmorrhage and liability to hæmorrhage in the brain. The headache is wonderfully benefited by a few leeches on the temples, but for the convulsions and more serious uræmic symptoms no remedy seems to be so efficacious as bleeding. Purging, diaphoretics, the cautious use of hot air baths and the

subcutaneous injection of pilocarpin may all be employed with advantage, but they all act better in severe cases after venesection has been performed.

(7) The pain, which is one of the most constant symptoms of thoracic *aneurysm*, is more often relieved by moderate venesection than by any other treatment. This was admitted by the late Dr. Hughes Bennett in the midst of his opposition to bleeding for pneumonia.* I shall never forget the case of a man who, for more than a week, had not been able to lie down, or enjoy more than broken and uninterrupted sleep. I found him the day after his admission to the hospital sitting up in bed, exhausted by want of rest and continually dropping asleep, but wakened again by the terrible pain he endured. The house physician had given him morphia in full doses without any benefit. He was bled from the arm to 6 oz., and before the blood ceased flowing expressed the relief he felt, and had fallen fast asleep on his pillow before the arm was bound up. His distress was never suffered to reach so advanced a point, and again and again on its return he was relieved by a similar or a still smaller abstraction of blood.

Bleeding from the arm is, in these cases, far more efficacious than the application of leeches, as well as more rapid in its effect. The object of the small venesections thus indicated is quite different from the full and repeated bleeding of Valsalva's method of treatment. The object is not to cure the disease, but to relieve its most distressing symptom. It must, we suppose, act by lowering the blood pressure in the aneurysmal sac. From experience of the manometer upon animals one would have supposed that the effects of a moderate abstraction of blood would be too transitory to be of practical service. But experience shows that this is not the case.

(8) The most frequent indication for blood-letting is general venous congestion with arterial anæmia indicated by *cyanosis* with dyspnœa, turgid veins, swollen liver,

* 'Principles and Practice of Medicine,' 3rd ed., 1859, p. 296.

albuminuria, pulsation in the jugular veins, and at the epigastrium, functional incompetence of the tricuspid valve (sometimes indicated by a systolic murmur), and a weak, small, and fluttering radial pulse. Under these conditions a moderate abstraction of blood relieves the distended right side of the heart and gives it a chance of recovering its propulsive power over the blood which reaches its cavities. The effects are no doubt transitory; but so, we may hope, is the condition we are seeking to remedy. The tendency to death is imminent and unmistakable; by obviating it we may often save the patient for the time being and give him a chance of recovery. However produced, the well-marked clinical condition just described appears to call for blood-letting as the most rapid and efficient remedy.

Where there is already anasarca, much less benefit is, I think, to be obtained than at an earlier stage. Again in valvular disease of the heart the effects are less striking and permanent than when the obstruction to the circulation is in the pulmonary capillaries; and the effect is better in cases of chronic bronchitis and emphysema than in those of lobar pneumonia, or of miliary tuberculosis. In that very grave combination of pericarditis with pleurisy and pneumonia, venesection may afford relief; but where there is extensive pleuritic effusion paracentesis is probably a more effectual and safer remedy; and where the pericarditis has gone on to the distension of the sac tapping the pericardium is the only effectual treatment. In cases of adherent pericardium, with or without valvular lesions, venesection has its place, and was remarkably beneficial in the case of a child recently under my care who suffered greatly from this very unfavourable condition; but the result can only be temporary.

The amount of blood to be withdrawn will differ with the object. To relieve the pain of aneurysm a very few ounces suffice,—3 to 6 is often enough, but it may be repeated whenever the pain returns. In cases of uræmic

convulsions bleeding should be more free and is often usefully supplemented by leeches. In a condition of cyanosis still larger depletion may sometimes be needful —from 10 or 12 to 15 ounces. In all cases the bleeding is most useful as an introduction to subsequent treatment. Purging is of course indicated in uræmia, aided by pilocarpin and other diaphorêtics ; and stimulation by ammonia or ether, with digitalis, senega or squills, in cases of bronchitis and of cardiac disease. In cyanosis, brandy as well as ammonia is often essential ; nor is there any contradiction in first lightening the heart of its load and then stimulating it to increased exertion.

Cases of Venesection, 1875—1890.

1. John Ward, bed No. 1, April, 1875, male, æt. 60. Chronic bronchitis, cyanosis, no cardiac disease, no albuminuria, respiration 62, almost suffocated. V.s. ad ℥x : much relieved, respiration sank from 62 to 50, but the patient died that night. There were pleural adhesions on both sides, emphysema, dilated and hypertrophied right side of the heart ; valves competent, kidneys normal.

2. John, 5, April 12th, 1875, M., æt. 25. A stout, healthy young man, not a drunkard. Acute lobar pneumonia of left base. On the third day, very ill with dyspnœa and pain. V.s. ad ℥x : no marked relief, although the pulse-tracing showed diminished tension. Next day profuse frothy expectoration, with signs of bronchitis on both sides ; treated with brandy and ammonia ; but he died that night. There was found grey hepatisation of the whole of the lower lobe of the left lung, and two small inflammatory patches in the right, with œdema and profuse bronchial secretion in both.

3. Mary, 15, December, 1875, F. Capillary bronchitis, with intense cyanosis, no orthopnœa. V.s. ad ℥viij. This was repeated, but the patient died.

4. Mary, 5, August, 1876, F., æt. 13. Mitral imperfection. V.s. repeated. (She was a patient of Dr. Wilks, in whose absence I ordered the first v.s.; after his return he ordered the second.) Improved and went out pretty well.

5. Stephen, 50, November, 1876, M., æt. 25. Pericarditis with endocarditis of the mitral valve, considerable hæmoptysis, and pleurisy, in course of rheumatic fever: third attack of rheumatism, the first four years ago, then three months ago; present attack slight. V.s.: blistered and given digitalis, ammonia, ether, and brandy. Died. Seventeen ounces of purulent serum in pericardium with shaggy lymph. Old valvular disease.

6. Mary, 7, November, 1876, F., æt. 29. Chronic tubal nephritis after scarlet fever, dropsy, pleurisy, uræmia, vomiting, eclampsia. V.s. twice, each time with decided improvement. After death the kidneys were found large, yellow, and mottled, with irregular surface and wasted cortex. Heart 15 oz. from hypertrophy of left ventricle. Numerous spots of hæmorrhage in retina.

7. John, 16, August, 1877, M., æt. 40, soldier. Enormous aneurysm of ascending aorta distending the right breast. Death from hæmorrhage into subcutaneous tissues. V.s. ordered, but scarcely enough blood withdrawn to have any effect.

8. Stæphen, 26, November, 1877, M., æt. 15. Aortic valvular disease, great pain and dyspnœa. Six leeches on chest and v.s. ad ℥iv. He was much relieved by the leeches. Died a few days later; moderate dropsy; aortic incompetence; bovine heart.

9. Mary, 6, August, 1878, F. Aortic obstruction and regurgitation; dropsy; purpura (probably secondary); extremely ill. V.s. ad ℥vj. Great relief. Went out much better.

10. Stephen, 31, August, 1879, M., æt. 30. Mitral disease after rheumatism. Anasarca. V.s. ad ℥iv ; great relief. Died some weeks later from acute pleurisy.

11. Stephen, 25, November, 1875, M., æt. 45. Aneurysm of abdominal aorta. V.s. ad ℥iij. Went out relieved.

12. Stephen, 32, November, 1879, M. Acute bronchitis with right pleural effusion, following an attack of delirium tremens ; great cyanosis. V.s. ad ℥viij. The pulse afterwards was irregular, and the cyanosis was not diminished ; he remained insensible, with increasing asphyxia, and died on the third day after the bleeding. Healthy kidneys; wasted brain ; thickened meninges ; moderate pleuritic effusion.

13. John, 5, April, 1883, M., æt. 43. Aneurysm of aorta ; severe bronchitis. Ice bag on tumour. Increased dyspnœa. V.s. ad ℥vj ; relief. Subsequently the aneurysm perforated the chest wall, and he died several weeks after the bleeding from dyspnœa with failing heart. There was no rupture of the sac, but an abscess had formed close to it, and had received pulsation from it during life. This was due to caries of the perforated sternum.

14. John, 2, June, 1883, M., æt. 60. Severe eclampsia with cyanosis. There was no evidence of renal disease. V.s. ad ℥xij. He recovered, and went out pretty well.

15. June, 1883, M., æt. 50. Excessive dyspnœa with pneumonia, bronchitis, and cyanosis. Attempted v.s., but only an ounce of blood could be obtained, and he died within an hour. Beside right lobar pneumonia with pleuritic effusion, there was general bronchitis with emphysema and ulcerative endocarditis. Rheumatism ten years before. Urate of soda in great toe-joint.

16. July 31st, 1883, M., æt. 35. Brought in from the

street in an apoplectic fit; insensible with stertorous breathing. V.s. ad ℥iv. The pulse quickened, but otherwise no change. Died two hours after admission. Extensive hæmorrhage in the left lenticulus, breaking into the ventricle and filling the third, fourth, and aqueduct.

17. Stephen, 8, August, 1883, M., æt. 40. Aneurysm of ascending aorta; bronchitis. Dry cupping between the shoulders. V.s. ad ℥xij. Fits of dyspnœa continued to occur, and one night laryngotomy was performed. Death, probably from hæmorrhage into the trachea. A large aneurysm was found pressing on the left bronchus and trachea.

18. Stephen, 2, October, 1883, M., æt. 36. Aneurysm of ascending aorta and innominate. V.s. ad ℥xv. Great relief, and smaller bleedings than this were repeated and found more beneficial than morphia, leeches or cupping in relieving the severe pain. "He had lost 132 oz. of blood by twelve bleedings in the course of a fortnight, and again and again had been thus rescued from impending death by suffocation."

19. Phil., 26, November, 1883, M., æt. 27. Acute tubal nephritis; uræmia with scanty urine and vomiting. Hot air baths, pilocarpin, purging and cupping were all tried, without relief of the uræmic symptoms. V.s. ad ℥viij was followed by some relief. Pericarditis came on with double pleural and peritoneal effusion and almost complete suppression of urine. After death, the kidneys weighed 22 oz., large and mottled.

20. March 11th, 1884, M., æt. 42. Drunkard; brought in insensible, without stertor, apparently from apoplexy. V.s. ad ℥vj, and turpentine enema. No improvement. Cheyne-Stokes' respiration. Temperature rose. Death from dyspnœa. Extensive hæmorrhage in the right corona radiata, not touching the surface or ventricles.

21. Miriam, 28, March, 1884, F., æt. 50. Chronic osteo-arthritis with disease of left apex, severe capillary bronchitis with ascites and impending suffocation. V.s. ad ℥xx. Relieved and apparently rescued from death. Died three days later. Chronic cirrhosis of one lung with adherent pericardium.

22. Philip, 37, July, 1884, M., æt. 28. Right pneumonia with pleuritic effusion. Excessive dyspnœa, impending suffocation. V.s. ad ℥vj. Relief. The pleuritic effusion afterwards increased, and 55 oz. of serum were drawn off by paracentesis. Recovery.

23. Mary, 33, November, 1884, F., æt. 40. Morb. Brightii. Eclampsia. V.s. ad ℥vij. Vomiting and convulsions ceased, but coma afterwards came on and she died without fresh convulsions. Granular kidneys; hypertrophied left ventricle with fibrous patches.

24. Phil., 33, August 1st, 1885, M., æt. 35. Morb. Brightii. Uræmia. V.s. ad ℥xviij. The convulsions were relieved, but he died in the same month. Large, smooth, white kidneys. Great hypertrophy of left ventricle.

25. Phil., 30, December 15th, 1885. Enormously fat man about forty years old. Severe bronchitis with dilated right heart, dropsy, and cyanosis. V.s. repeated with benefit, but died ten days after admission. Bronchitis and pulmonary œdema, with dilated right side of heart.

26. John, 1, January, 1886, M. Morb. Brightii with left pleurisy, cystitis from stricture, and uræmic symptoms. V.s. ad ℥iv. Relief. Steady improvement, and went home much better.

27. John, 5, January, 1886, M., æt. 21. Acute capillary bronchitis following previous attacks, with emphy-

sema. V.s. twice, but the relief only temporary. After death there was extensive emphysema with dilatation of the right side of the heart. A well-nourished, muscular young man, a stonemason, who had had winter cough for several years; he was working up to a week before his death.

28. John, 11, January, 1886, æt. 41. Chronic morb. Brightii with uræmia, delirium and excessive dyspnœa. V.s. ad ℥viij and leeches to sternum. Pilocarpin. In great danger for about a week, but then improved greatly. He died at last, however, two months later. Granular contracted kidneys with hypertrophied left ventricle, and lithate of soda in the joints were found after death.

29. Mary, 31, March, 1886, F., æt. 40. Morb. Brightii, uræmia, pericarditis, and retinal hæmorrhage. V.s., little benefit. After death, contracted granular kidneys, hypertrophied left ventricle, pericarditis, and œdema of the lungs were found.

30. Phil., 22, June, 1886, M., æt. 38. M. cordis with albuminuria and orthopnœa. V.s. ad ℥iv. The relief was but slight, and he died after about a month. The heart weighed 21 oz., but there was no valvular lesion.

31. Phil., 36, August, 1886, M., æt. 25. M. cordis: aortic regurgitation and mitral obstruction with regurgitation. V.s. ad ℥iij. Marked relief when almost moribund, and he went out after some months much relieved. Readmitted the following year and again relieved by bleeding and by leeches on more than one occasion. At last became delirious and died.

32. Mary, 24, August, 1886, F., æt. 16. Acute Bright's disease with dropsy, uræmia, eclampsia. V.s. ad ℥iv, pilocarpin, hot air baths. Remarkable improvement,

uræmic symptoms ceased, and she went out apparently well.

33. Phil., 24, an elderly man, October, 1886. Morb. cordis (mitral incompetence) with bronchitis, albuminuria and jaundice, delirium, great dyspnœa, and impending suffocation. Twice saved by v.s., the first time 18 and the second 12 oz. were withdrawn. At last he died, and beside cardiac disease and bronchitis, cystic kidneys were found.

34. Miriam, 27, November, 1886, F., æt. 74. Acute bronchitis with cyanosis threatening suffocation. V.s. ad ℥viij. Decided improvement, but relapse and death some days after. Beside bronchitis she had granular kidneys with numerous minute cysts.

35. December, 1886, M., æt. 21. Left pleurisy with effusion, 8½ oz. removed by paracentesis, still dyspnœa severe, dulness at both bases with irregular pulse. Only a week ill. On the third day v.s. ad ℥iv, followed by little or no relief. After death, beside double pleuritic effusion, there was extensive lobular pneumonia and acute pericarditis.

36. Phil., 25, January, 1887, M., æt. 50. Acute bronchitis, urgent dyspnœa. V.s. ad ℥vj. No relief; died soon after admission.

37. March 3rd, 1887, M., æt. 50. M. Br. uræmia. Very stout, intemperate, and dropsical. Walked into the hospital to ask for a dose of physic; became rapidly ill, and the same afternoon was found pallid and insensible, with small hard pulse, and Cheyne-Stokes' respiration. V.s. ad ℥iv, and turpentine enema. He died the same evening, and there was found, as expected, granular and cystic kidneys, with hypertrophied left ventricle, anasarca, and some effusion in each pleura.

38. Phil., 24, August, 1887. A very stout man. Great dyspnœa, with bronchitis, albuminuria, and gout. V.s. ad ℥v: relieved, and went out, after several weeks, apparently well.

39. Phil., 33, November, 1887, M., æt. 52. Admitted with bronchitis and albuminuria. Suddenly worse, with râles over both lungs, and hæmoptysis, apparently moribund. V.s. ad ℥viij, followed by subcutaneous injection of brandy, and a brisk emetic. Revived, continued very ill with hæmoptysis, gradually improved, and at last went out well.

40. Phil., 37, August, 1888, M., æt. 54. Morb. Brightii. Sudden attack of pulmonary œdema, with epistaxis and intestinal hæmorrhage. V.s. ad ℥x: no marked improvement. Death two days after. Large white kidneys beginning to undergo granular contraction; œdema of lungs and larynx; no cardiac hypertrophy.

41. John, 6, November, 1888, M., æt. 18. Admitted with intense dyspnœa and cyanosis. Acute tuberculosis, diagnosed as capillary bronchitis from miliary tubercles. V.s. ad ℥iv: very slight relief, and death with cyanosis. Both lungs found full of miliary tubercles; no chronic phthisis; tubercles in liver and kidneys, and in peritoneum; a few tubercular ulcers in ileum. No caseous material or primary seat of tubercle discovered.

42. Phil., 24, March, 1889, M., æt. 29. Hard drinker; subject to winter cough. Admitted with intense dyspnœa, orthopnœa, and cyanosis; diagnosed as acute capillary bronchitis with dilated right heart and tricuspid regurgitation. V.s. ad ℥xij; calomel, ammonia, and brandy; six leeches to chest. Relief after the bleeding. Died thirty-six hours after admission. Emphysema, with œdema of lungs and pleuritic adhesions, lobular pneumonia, dilated right heart, and nutmeg liver; kidneys normal.

43. Phil., 38, October, 1889, M., æt. 8. Severe and advanced mitral disease and adherent pericardium, without dropsy; great orthopnœa, pain, and distress. V.s. ad ʒiss. "This did great good, and he slept that night instead of kneeling up in distress. He took food again, was dressed, and sat up in a chair for several days. Then a fog came on, and he died after a few hours. The house physician had again tried to relieve him by opening a vein. The pericardium was found universally adherent, with incompetent aortic valves and puckered and dilated mitral."

44. December, 1889, M., æt. 24. Admitted with intense dyspnœa and cyanosis. Treated by an emetic, and v.s. ad ʒvj. Somewhat relieved, but died on the third day. Emphysematous lungs with bronchitis and lobar pneumonia; dilated right side of heart.

45. Philip, 24, January, 1890, M., æt. 44. Beer drinker; subject to winter cough. Acute bronchitis with lobular pneumonia; clubbed nails; cyanosis and delirium. (A history of hæmoptysis led me to suspect chronic phthisis.) V.s. ad ʒvj. Decided relief was obtained, but he afterwards sank. Emphysema, bronchitis, and extensive lobular pneumonia. Recent acute double pleurisy. Right side of heart not dilated. Kidneys somewhat granular, with the cortex of one decidedly wasted.

46. Philip, 36, April, 1890, M., æt. 33. No history of rheumatism; maculæ from lues; aortic obstruction and regurgitation; pallor; pain; orthopnœa. V.s. ad ʒvj: relief. Died next day. Fungating disease of both aortic and mitral valves, with renal embolism. Cardiac organs: no atheroma. Only ill since Christmas.

47. Philip, 33, September, 1890, M., æt. 40. Bronchitis and emphysema. Dilated heart. Feeble pulse. Scanty high-coloured urine without albumen. Very ill. V.s. ad ʒvj: marked benefit. Steadily improved and went out well.

48. Philip, October, 1890, M., æt. 38. Aneurysm of transverse aorta. Great distress. Small v.s. ineffectual. Went out unrelieved.

49. Philip, 39, October, 1890, M., æt. 8. Adherent pericardium and mitral regurgitation. V.s. ad ℥iv: marked relief. Death later.

Summary of Cases.

Bronchitis and cyanosis		13
Acute capillary bronchitis, with emphysema, bronchial pneumonia, or, in one case, miliary tubercle.		
Lobar pneumonia and cyanosis		4
Morbus cordis, aortic, mitral, or in one case idiopathic hypertrophy, with cyanosis		8
Pericarditis (one with pleurisy and lobular pneumonia) .	2	4
Adherent pericardium (one with chronic cirrhosis, one with mitral regurgitation)	2	
Morbus Brightii (with anæmia)		11
Aneurysm of aorta		6
Apoplexy (cerebral hæmorrhage)	2	3
Epilepsy (or eclampsia not due to uræmia) . .	1	
		49

Summary of Conclusions.

A. *Bleeding contra-indicated.*

For pyrexia, enteric fever, rheumatism, hyperpyrexia.

For frequent and bounding pulse.

To cut short pneumonia, pleurisy, pericarditis or peritonitis, meningitis or acute nephritis.

To relieve "cerebral congestion," apoplexy, &c.

To stop hæmorrhage.

B. *Indicated.*

I. To relieve the right side of heart in pulmonary obstruction, shown by cyanosis, pallor, dyspnœa, and a small feeble pulse.

1. In chronic bronchitis and emphysema, acute capillary bronchitis, lobular bronchial pneumonia, acute tuberculosis.
2. In pneumonia, with much cyanosis.
3. In cardiac disease, with great dyspnœa.

4. From pericarditis, with great effusion, orthopnœa, and feeble or irregular pulse.
5. From adherent pericardium, with or without valvular lesions.

II. To reduce arterial tension, and relieve pain due thereto.
1. In aneurysm.
2. In aortic valvular disease, with high tension and headache.

III. 1. To relieve uræmia, in acute nephritis with suppression, or in very chronic nephritis with headache and eclampsia.
2. In epilepsy (not of uræmic origin) and apoplectiform eclampsia.

Immediate indications.—1. Excessive cyanosis, with or without dropsy, whether pulmonary or cardiac in origin, with full veins and empty arteries, pallor, dyspnœa, orthopnœa, asystolian, small, feeble, irregular, or imperceptible pulse. Perhaps repeat several times.

2. Uræmia, with hard pulse, headache, and especially with convulsions and hæmorrhage.

3. Intense pain of aneurysm, and some forms of aortic and valvular disease.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 48.)

MACRODACTYLY,

AND SOME OTHER FORMS OF CONGENITAL OVER-GROWTH,

AND THEIR RELATION TO TUMOURS.

BY

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In the Pathological Museum of the University of Cambridge are the six following specimens and casts.

1. The hypertrophied second toe amputated from a child, æt. 4, by Mr. Morris, of Bishop's Stortford. At birth it was thrice the size of the great toe, and grew more rapidly than the other parts. The phalanges are of great length, but not otherwise diseased. The tendons are proportionately large, and there is a superabundance of skin and adipose tissue all round.

2. A cast presented by Mr. Cathcart, of Edinburgh, of the giant middle finger of a child æt. 20 months. The condition was congenital; but the finger had grown out of proportion to the other fingers, and was amputated by Mr. Joseph Bell.

3. A cast presented by Mr. Cathcart of the great toe of a girl, æt. 13. The affection was congenital. The second toe had still larger proportions, and was amputated in childhood.

4. A cast, taken and presented by G. May, of Trinity College, of the left foot of a woman, æt. 45, in the London Hospital. The two inner toes and the inner side of the foot hypertrophied from birth.

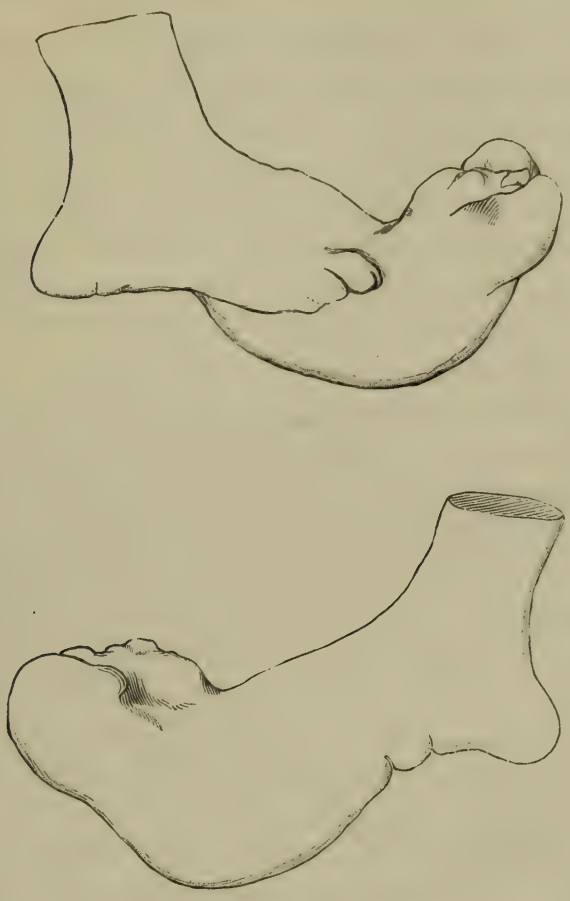
	Left foot.		Right foot.
The inner border of the foot from the point of the heel to the end of the great toe . .	13½ in.	...	10 in.
Circumference of great toe near the end .	7	...	2½
„ of second toe „ .	5	...	1½
Outer border of foot	7	...	7

The sole is flat, and bulges on the inner side as in "flat-foot." No similar condition in any member of her family or any of her children.

5. A cast of congenital hypertrophy of the fore and middle fingers of the right hand of a boy æt. 12. The forefinger measures from the metacarpal bone four inches and a half, the second and third phalanges measuring two inches and three quarters, circumference of thickest part three inches and three quarters. The middle finger measures three inches and three quarters in length, in circumference three inches, and is remarkably deflected towards the ulnar side in the second and third phalanges. The thumb and other fingers normal. The cast was presented by Dr. Walker of Peterborough, who amputated the two hypertrophied fingers because they were stiff, much in the way, and a source of mortification to the boy. The growth was not disproportionate to that of the other fingers. There was no similar condition in any member of the family.

6. The cast of the foot of a lad, æt. 9, whom I saw on the beach at Cromer in 1837, and whom I took to the Norwich Hospital, where he was under the care of Mr. Crosse, whose apprenticed pupil I then was. The enlargement, as seen in the accompanying rough sketch, was great, and affected the three inner toes of the right foot.

It was congenital, and proceeded with a rapidity out of proportion to the general growth ; and it was extending upon



the sole more manifestly than in any other direction. The two outer toes were rather smaller than the corresponding toes of the other foot. The mass appeared to be chiefly composed of fat covered with rather coarse skin, and the nails of the hallux and second toe were much larger than natural. The affected toes were much extended, and the extensor tendons tight. No tenderness or pain. The muscles of the leg were enlarged, and the lad could run about as well as other boys. His father, mother, brothers, and sisters were fine, well-made persons. The hypertrophied parts, being unsightly, cumbrous, and increasing,

were removed by Mr. Crosse, the ends of the enlarged metatarsal bones being taken away, and sufficient skin saved from the dorsum of the foot to cover the sole and leave a good useful foot with little deformity or inconvenience. It was necessary to dissect out the fatty substance which extended up between the metatarsal bones. The mass was composed mainly of adipose substance. The bones were enlarged, but not otherwise diseased. The specimen, from which, however, the bones have been removed, is in the museum of the Norwich Hospital; and Mr. Thomas Crosse, the indefatigable curator of the museum, was good enough to take it out of the bottle the other day and verify my recollections of it.

Curling[1] records the case of a girl (No. 7) in whom the fore, middle, and ring fingers of the right hand, and the thumb, index, and middle fingers of the left hand were greatly hypertrophied, all the parts—bones, articulations, integuments, and nails—being developed in excess. The middle finger of the left hand was remarkably curved outwards (to the ulnar side), this having been "occasioned apparently by a displacement of the extensor tendon which forms a bridle along the outer edge;" the movements of the fingers but little impaired. The fingers felt cold. Pulsation in the digital arteries indistinct. The fingers were remarkably large at birth, and had grown out of proportion to the rest of the body. He mentions the following: (No. 8.) A child aged two, seen by Professor Owen, with the middle finger of each hand twice as long and more than twice as thick as the index. (No. 9.) A cast, with particulars by Sir James Paget, of the right hand of a man, aged fifty, with hypertrophied first and second fingers; the second was of enormous size, and curved outwards as in No. 7. The parts bore the same proportion as at birth, and were not the cause of any inconvenience. (No. 10.) The cast of the hand of an adult in the museum of King's College, with the middle finger congenitally hypertrophied. The hands of other members of the

[1] 'Medico-Chirurgical Transactions,' xxviii, 337.

family are reported to have been deformed in a similar manner. (No. 11.) The case of a girl, aged five, by Mr. Power, of Dublin,[1] with the middle finger of the right hand much, and the index and ring finger somewhat enlarged, particularly the ring finger, which was divaricated from the middle in consequence of its abnormal growth. (No. 12.) Dr. John Reid[2] gives the case of a boy, aged thirteen, in whom there was increased nutrition of the thumb and first finger of the left hand. The enlargement was associated with enlargement of the radial artery and elevation of temperature, was observed at birth, and continued to increase. In this paper Dr. Reid also describes the case (No. 13) of a lad, aged fifteen, with congenital hypertrophy of the whole of the left upper limb including the scapula; and (No. 14) that of a girl, aged two, with congenital hypertrophy of the middle toe which equalled in bulk all the remaining four toes, the phalanges and metatarsal bone being of great size.

Busch, in an excellent article,[3] relates two cases. One (No. 15) a man, aged twenty, with congenital hypertrophy and deformity of the three inner toes, which were much over-extended, and the adjacent part of the sole of the right foot, the growth proceeding out of proportion to the rest of the body and advancing along the sole (resembling, in short, No. 6). Pirogoff's amputation was performed. The phalanges, metatarsals, and inner tarsal bones were enlarged, especially at their articular ends; and these were deformed, tuberculated, and in some instances ankylosed; the ligaments thick, and the synovial fringes pronounced; much and disproportionate increase of fatty tissue, causing deformity and bulk; the arteries and nerves unaltered; the dorsal veins in part greatly thickened by fibrous formation in concentric sheets in their walls without increase of lumen; two outer toes quite free from hypertrophic change; the small muscles

[1] 'Dublin Journal of Med. Science,' xvii, 244.

[2] 'London and Edin. Monthly Journal of Med. Sc.,' 1843, p. 198.

[3] Langenbeck's 'Archiv für klinische Chirurgie,' vii, 174, and pl. vi.

of the foot thin and pale, but the tendons passing from the leg, especially that of the tibialis posticus, very thick ; two small fat swellings in front of the tibia ; the tibia and fibula rather larger but not thicker than those of the other leg ; the thighs of equal length.

Busch's second case (No. 16) was a girl, aged twelve, with hypertrophic condition of the second and third toes of the right foot, which were over-extended and projected beyond the other toes. Being inconvenient, unsightly, and disproportionately increasing, they were removed, and the projecting end of the second metacarpal was removed also. The hypertrophy of the bones affected chiefly their ends (the epiphyses). The capsular ligaments were very thick, and contained cartilage nodules near their attachments. The joints were in other respects normal, as also were the arteries, veins, nerves, and tendons. The fatty tissue on the dorsal as well as on the plantar aspect was much increased.

Busch quotes a case (No. 17, from Graefe[1]) where Klein amputated an hypertrophied forefinger of the left hand (sex and age not given), the second and third phalanges of which projected laterally over the other fingers. No vessel required ligature at the time, though there was some bleeding afterwards ; a case (No. 18) by Guersant[2] of hypertrophy of the fourth and fifth fingers ; a case from Böhm[3] (No. 19) of a girl, aged sixteen, in whom the second toe of the left foot was three times as long and twice as thick as that of the other foot. The examination of the part when amputated showed nothing abnormal in the arteries and nerves. The plantar fat-pads were large, and the ligaments thick. (No. 20.) Wagner[4] describes the case of a lad (aged eighteen) in whom all the right hand (except the thumb, which was smaller than that of the left hand) and the forearm were hypertrophied, and grew out

[1] From Graefe and Walther's Journal, vol. vi.

[2] 'Gazette des Hôpitaux,' 1857, p. 463.

[3] 'Inaugural-dissertation über Macrodactylie,' Giessen, 1856.

[4] 'Schmidt's Jahrbücher,' iii, Supplementband, 1842, s. 66.

of proportion to the rest of the body. The end of the forefinger, which was small, was over-extended. The size was apparently due chiefly to fat-growths. There was also a fat-growth about the right breast extending from the sternum to the axilla, which had begun when he was five years old. (No. 21.) Legendre, quoted by Böhm, saw a child, aged four and a half, with hypertrophy of the fourth and fifth fingers and the ulnar side of the hand, chiefly on the palmar aspect. The fourth finger was bent backwards and to the radial side in consequence of inequality of growth of the two sides. (No. 22.) Ideler (in seiner 'Dissertation,' Berlin, 1855) gives the case of a lad, aged twelve, in whom there was hypertrophy of the three middle toes, including the metatarsal bones, of both feet, with lipomatous condition of the toes and soles and somewhat of the dorsa; several small lipomata in the left leg, and a large congenital lipoma in the left buttock. In consequence of the increasing deformity the foreparts of both feet were removed by amputation, and the metatarsals and phalanges of the three middle toes were of abnormal length. The other toes appear to have been rather undersized. (No. 23.) Wulff[1] gives the case of a man, aged thirty-two, in whose right hand the first three fingers (thumb, index and middle fingers) were congenitally hypertrophied. The growth had continued *pari passu* with that of the rest of the body, but the increase had been greater in the last few years. There was much enlargement of the bones, especially in the neighbourhood of the metacarpo-phalangeal joint of the thumb, the terminal joint of the index, and both the phalangeal joints of the middle finger; external bending of the fingers and great increase of the fat-tissue. The arteries were apparently alike in the two limbs. (No. 24.) Burow[2] mentions a Polish girl, aged six, with hypertrophy of the second and third toes with their metatarsals. The increase was proceeding quickly, and the parts were removed. (No. 25.)

[1] 'Petersburger med. Zeitschrift,' 1861, 10 Heft, s. 281.

[2] 'Deutsche Klinik,' 1864, No. 27.

Mr. Holmes[1] represents the cast of the left foot and leg of a child, aged twenty months, affected with congenital enlargement of the foot and leg, which was so inconvenient and on the increase that amputation in the leg was performed, under the impression, he believes, that the disease was of malignant nature. Anatomical examination, however, showed that the bulk was due to an unusual deposition of fat and cellular tissue, the muscles as well as the bones being normal.

It appears from the above-recited cases (more might doubtless be collected) that the parts more frequently affected with this congenital "overgrowth," "hyperplasia," "hypertrophy," or "macrodactyly," are the digits on the inner—the radial and tibial—sides of the hands and feet, more particularly the second and third digit in the hand, and the first, second, and third in the foot; though in some cases (Nos. 20 and 21) the other digits were affected. In Nos. 6 and 22 the unaffected digits were somewhat undersized. The adipose tissue, the bones, and the skin[2] participated in the overgrowth, and usually in a proportionate degree; though in some the adipose tissue was increased to a disproportionate extent. The ligaments, the synovial fringes of the joints, and the tendons were in some observed to be enlarged. In No. 15 there was marked increase of the fibrous tissue in the coat of one of the veins. In this case also the articular ends of the bones were nodulated and deformed, and in one joint ankylosed, the parts thus presenting a resemblance to the condition often found in rheumatic arthritis, and to that sometimes observed in the acromegaly of adults. In the hand the condition was mostly limited to the digits, though in No. 13 the whole upper limb was involved; but in the foot it extended beyond the digits in several instances, the metatarsal bones

[1] 'System of Surgery,' iii, 798.

[2] I do not find it observed that the cutis or the cuticle was very thick, or the hair long or superabundant, in any of the cases, as is so often seen in "moles." The large size of the nails was a consequence of and proportionate to the large matrix from which they grew.

of the foot and even the bones of the leg (No. 15) being enlarged. The affection showed an especial tendency to spread along the sole of the foot, and the adipose tissue to grow up between the metatarsal bones.

The various tissues, except in No. 15, were simply hypertrophied (overgrown), but not otherwise diseased; and no abnormal condition of the blood-vessels, save the thickening of the veins in No. 15, or of the nerves was discovered in any case. The over-extension of the toes noted in some was probably caused by the adipose growth on the plantar aspect exceeding that on the dorsal; and the lateral curvature observed in the fingers was probably due to an irregularity in the growth on their two sides rather than, as suggested by Curling, to the influence of the extensor tendons.

In Nos. 7, 8, and 22 the condition was symmetrical or nearly so. It occurred about equally in boys and girls, in the hands rather more often than in the feet.

This sort of congenital overgrowth, attended usually with a relatively proportionate increase of the several tissues concerned, is not confined to the hands and feet. It occurs also in the face and other parts. I have recorded[1] the case of a girl, aged twelve, in whom there was congenital overgrowth or hypertrophy of the gums on the left side, without and within the alveolar border, which had increased so much as to bulge the cheek and project the lips, and cause such inconvenience as to render an operation for removal of the deformity necessary; the teeth, though in great measure covered by the mass, and the maxillary bone were normal. The lips on the left side were larger and thicker than natural, also the left ala of the nostril, and the left eyelids and eyelashes, and the hair of the eyebrow and that of the forehead, which latter descended lower than on the right side. The pinna of the left ear was a quarter of an inch longer and a little thicker than that of the right ear. The left tonsil and side of the palate and the papillæ on the left side of the

[1] 'Annals of Surgery,' iii, 1.

tongue also were larger than on the right side. I refer in that paper to other more or less similar cases. In my case the continuance of disproportionate increase was, I believe, confined to the gums. Instances of congenital hypertrophy of the lips have been observed by others;[1] and the cases of congenital hypertrophy of the tongue, in which all the tissues, some more and some less, are increased, are of the like nature.[2]

The real interest of these cases lies in their pathology, and its bearing upon other pathological conditions. They obviously consist in an excess, an abnormally excessive growth of a normal part of the body—an excess not depending upon any superabundance of nutritive supply or any modification of nerve-influence, but upon an excess, a want of due restraint, of that developmental force by which the several organs and structures acquire and maintain their proper dimensions and relations to one another, and by which their relative growth at different periods of life and under different circumstances (as of the genital organs at puberty) is determined. The nature and essence, and habitat or source of this force is a mystery, perhaps past finding out. It seems to be shared, as an inherent quality, by each part and by each tissue in the same manner as by the primitive germ, and as a derivative from it. Each part forms and grows by the *vis inertiæ* in itself, though for the supply of the material by means of which it forms and grows it is dependent upon external sources, as the blood in the vessels, and for its co-operation with other parts upon nerve-influence; just as each soldier in a regiment has his own independent force and activity, but depends upon the

[1] Ashurst's 'International Encyclopædia of Surgery,' v, 463.

[2] Now and then a similar overgrowth commences later in life, as in the case of a woman, aged thirty, mentioned by Cohnheim ('General Pathology,' ii, 750), in whom hypertrophy of both hands and the lower part of the face thus took place. It seems probable, however, that this should be regarded as a case of "acromegaly," which, I may observe, is a morbid condition occurring usually in adults and affecting the system generally, not, like "macrodactyly," a localised congenital overgrowth.

commissariat for his maintenance, and the trumpet call for harmonious co-operation with other members of his corps. He may of his own free action take an insubordinate line, to the detriment of his fellow-soldiers and the damage of the service; and he may involve others by his example and influence. The like insubordination to the controlling and harmonising forces of nutrition and growth may be supposed to occur in any tissue or at any period of life. It may continue or it may cease. It may remain localised or it may spread to surrounding parts. It may consist in, or lead to, a mere increase and maintenance of *normal* structure, or it may result in various degenerations or deviations from the normal. It may be attributable to an obvious local cause or stimulus, and may cease with that cause; but in many instances no such cause can be assigned.

Admitting the view thus taken as a working hypothesis, not regarding it as a full explanation, for that is unattainable in our ignorance of the nature of developmental and growing forces, but referring these macrodactyls and other abnormal growths to an over-exercise of that force by which each part attains and maintains its size and structure, we find, I think, a better idea and explanation, or theory, of the nature of tumours than that afforded by the "latent embryonic germ theory" propounded by Cohnheim, and too readily, as I believe, accepted by many modern pathologists. It seems, indeed, a piece of supererogation thus to increase our difficulties by resorting to this view, which has *two* postulates instead of one: *first*, the presence of the latent germs, of which I need scarcely say there is no evidence; and *secondly*, the starting of these germs into a mischievous or insubordinate activity without any assignable cause.

Let us trace this simple theory of "overgrowth" or "hyperplasia" which I am advocating, and have for years maintained and taught, as against the "latent germ theory" in its application to various growths.

The congenital MACRODACTYLS and other congenital

hypertrophies above described are obviously mere enlargements or overgrowths of normal tissues, of a finger, a toe, a lip, or a tongue, which in some instances proceed indefinitely, in some cease to increase after a time, in some extend to the surrounding tissues, and in some remain limited to the part first affected.

NÆVUS is a similar condition of blood-vessels, commencing usually a little before birth, and spreading for a time, but commonly for a limited time only.

MOLES or MOTHER-MARKS, in which the cutis is thick and pigmented, sometimes nævoid or tuberculated, and usually with overgrowth of hair, now and then continue to increase after birth, or start an increase at some later period, as in a case I lately saw where the tubercles grew out into masses requiring removal.

The other FŒTAL TUMOURS, consisting of cysts, *serous* or *dermoid*, of cartilage, bone, fat, or other structures (the so-called "parasites" are probably, for the most part, of this nature), often exhibit, as might be expected at the time of life when development is in high activity, an insubordination of developmental force as well as of growth, which leads to variation, it may be to undue exaltation, of structure, as well to increase of size, as evinced by the presence of cartilage, bone, skin, and even glandular structure. These tumours usually remain localised, and commonly, as exemplified by the supra-orbital dermoids, cease to grow after a time, their developmental and growing tendencies ceasing with the diminishing developmental and growing forces of the body. They are fully as well accounted for by an excessive growth and change of normally existing germs as by the presence of superabundant germs and an excessive growth of them. That such manifestations of variety and exaltation of structure should be observed in the ovary (they are probably congenital or occurring in early life) is in accordance with the marvellous formative properties of the Graafian bodies of that organ when normally acting under the influence of the natural stimulus.

With regard to the extra-uterine growths, a WART is a localised enlargement or overgrowth of the papillary tissue of the skin, continuous with the surrounding skin and spreading into it, the overgrowth being commonly excited by some local irritation. POLYPI are of like nature, not spreading so much into the mucous membrane with which they are continuous as growing in the direction of least resistance. The DIFFUSED LIPOMATA of the neck and the DIFFUSED MYOMATA of the uterus, PERIOSTEAL FIBROIDS, and EPULIS are clearly, like macrodactyls, localised overgrowths of the respective tissues, started into activity by some unknown cause. The continuity of these growths with the tissues in which they are found, and the manner in which they spread into these tissues by exciting the like overgrowth in them, seem sufficient evidence of their nature and derivation from normal structures, and to be incompatible with the idea of their derivation from latent and suddenly active superabundant germs.

The more CIRCUMSCRIBED TUMOURS are but a step removed from these last, in that the insubordination of growth is, and remains, more limited. Moreover, in some of these the limitation is not absolute—that is to say, the growth is not quite confined to the starting-point, but extends to a certain extent in the surrounding tissue. This, at least, we may infer to be the case in many of the subcutaneous lipomata from their lobulated character and the manner in which they are penetrated by fibrous septa passing into them from the skin, and giving the characteristic dimpling or fissuring of the surface. They are most frequent in the persons and parts where the growth of normal fat is greatest, and they sometimes take their start at many points. The MAMMARY ADENOMATA are also good illustrations of localised overgrowth of a normal structure, composed of modified mammary tissue, usually remaining continuous with the mammary gland, and apparently, in some cases, owing their increase partly to an extension to or an involving of it. The tendency to this insubordinate growth may exhibit itself at any part, giving rise to two

or three tumours in a mammary gland, to a dozen in a uterus, and to a hundred or more in the subcutaneous fat. They usually present the general structural features of the tissue in which they are found, though in some instances, as by the presence of cartilage in the testicle and the salivary glands, they show developmental variations from the normal connective tissue of the part.

The osseous system is fertile in an interesting and instructive gradation of these more or less insubordinate productions, ranging from the callus, often excessive after fracture, and the osteophytes about joints, ulcers, and necroses, through the varieties of scleroses and nodes, to the ossifications into tendons, exostoses, enchondromata, and osteomata. Some of these are the result of an increased supply of blood caused by congestion of blood-vessels from various causes, while others take place without any apparent cause, and can only be ascribed to an overgrowing of the cartilage or bone structure where they spring; and in proportion to their independence of any known cause are they resistent of any known treatment.[1]

The MALIGNANT GROWTHS fall into the same category. The insubordinate growth, set agoing often by some continued irritation, when once begun spreads along the adjacent tissue; and the pertinaciously multiplying germs of the tissue in which they form insinuate themselves into the surrounding structures, reaching the lymph- and blood-streams, and carrying their evil influences wherever they go. This is well shown by the carcinomata, the epithelial growths of which infect the surrounding epithelium, pass into the subjacent tissue, infiltrating it, partly destroying and replacing it, and partly giving rise to a low fibroid hyperplasia of its connective tissue, whereby is formed the areolar network in which the cancer-cells and germs lie

[1] The little pieces of cartilage occasionally found in the shafts of rickety bones, as the remnants from imperfect and irregular ossification at the epiphysial lines, do not really afford any argument in favour of the embryonic germ theory of tumours, forasmuch, as far as I am aware, they do not ever become developed into tumours.

nested. The functionless included products of these germs, being out of the range of normal metabolism and the natural outlets for the epithelium, die and decompose, and force an abnormal exit through the medium of ulceration or mortification, while the infiltrating and spreading products are carrying the like destructive influence to neighbouring and to distant parts.

Into the primal cause of tumours, that which first excites and gives continuance to the impulses of inordinate growth and assimilative power, we can, as I have already said, no more penetrate than we can into the primal cause and regulation of normal growth and nutrition. The gradations I have mentioned from callus to osteomata, and those between simple warts, warty epitheliomata, and infiltrating epitheliomata, and the known exciting causes of some of these, may help us to approach a somewhat nearer knowledge of the ætiology of the tumours, but a clear conception of it must await a closer insight into the laws of nutrition and growth. I wish here merely to accentuate the view that whatever may be their several exciting causes, and however much they may differ in their characters and progress, macrodactyls and nævi, warts and tumours, simple and malignant, are essentially alike in the feature of being overgrowths of the parts or tissues in which they take their origin.

I can quite agree with Cohnheim that there is "no fundamental difference between fœtal and extra-uterine growth," and that the real cause of a tumour may be "sought in a fault or irregularity in the embryonic rudiments." Indeed, some such faults or irregularities, evincing themselves in the course of the developmental progress of those rudiments, or rather of the structures resulting from them in any of the several stages from the fœtal to the senile state, may be regarded as the source of a large number of the weaknesses and maladies, tumours among others, which occur at various periods of life. They constitute, indeed, a widely embracing factor in pathology; and the body which is most free from such faults is least

prone to disease and best fitted to hold on through the greatest length of time.

Postscript.—Mr. William Anderson, in a valuable paper in 'St. Thomas's Hospital Reports,' vol. xi, gives a case of giant growth, apparently congenital, of the left lower limb, with tumour formation in connection with the superficial parts, in a woman æt. 25. The growth was progressive, and increased most rapidly after the ordinary body-growth had almost reached its complete development. Many cases of congenital hypertrophy are referred to, and much information on the subject given.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 60.)

TWO CASES OF IDIOGLOSSIA.

WITH PHONOGRAPHIC DEMONSTRATION OF THE PECULIARITY OF SPEECH.

BY

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AND

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Received December 3rd, 1890—Read March 10th, 1891.

The younger of the two cases which we show this evening to the Fellows of this Society was brought to one of us, in September, 1889, for an opinion as to his future education, it being believed by his friends that the defect in speech which we have to bring under notice to-night was a form of deaf mutism.

That this was not the case was at once apparent, the child's hearing, as well as his appreciation of what was said to him, being perfect; and he clearly was able to express himself in articulate sounds, which—and herein was the peculiarity of the case—it was impossible to understand.

The boy had recently been rejected by an asylum-school under the impression that he was dumb, and it was suggested that his was a suitable case for the Deaf and Dumb Asylum.

Although we show this evening two brothers, both similarly affected but in varying degree, there is also a sister, who is older than either of them, who is slightly affected in the same way.

These three children are the offspring of the same parents, who are English, without admixture of any foreign blood. They have never been used to hear a foreign language spoken. Their father is dead. He is said to have spoken badly and to have stammered; it is also said that he cured himself at the age of twenty by some American process. He died at fifty. His mother was in a lunatic asylum. A paternal uncle of these children is said to have talked indistinctly many years ago, but now he speaks distinctly. Their mother is a nervous emotional woman.

We will now describe each of these children in detail, taking first the youngest, Alfred, who is the most affected, and was the child first introduced to us. In our description of him we shall present to the Society a picture of the characteristics of this remarkable infirmity, hitherto unnamed, and as far as we are aware, but imperfectly studied.

Alfred James P— was first seen in September, 1889, at the age of nine years. He had been educated between the ages of five and nine in dames' schools, where he had acquired a character for sharpness and ability, and also an amount of learning reasonably proportionate to his age and station. The mistress of his school had, however, observed that his speech was incomprehensible, and she specially tried to improve his articulation, but failed to do so, and expressed her opinion to his friends that he was tongue-tied, or that his tongue was too short. His mother, on being questioned by us in regard to this matter, said she had observed the same peculiarity since he first began to speak, but she had not troubled herself about it. Both she and the mistress were convinced that he understood all that was said to him, but he failed to express himself in intelligible words to them and his juvenile companions.

When first seen by us the child was, as now, healthy, and except for his speech, perfectly normal. He is intelligent and well educated for his position ; he can do simple sums, read to himself, and write well from dictation. His orthography is correct. No physical defect is discoverable, and his tongue and larynx are normal in form and action. He is right-handed ; his viscera are not transposed ; there is no facial hemiatrophy ; and he does not stammer.

Directly he was spoken to it was evident that he was not like other children. His intonation and mode of articulation were perfect, but he employed sounds to express his words which were unlike those of English, or of any language known to us. If he were speaking at such a distance that one was not surprised at being unable to catch every word, his speech gave the impression, by the regular inflexion of his voice and the occasional repetition of the same sounds, that it was the speech of someone conversing intelligently in an unknown tongue. In close conversation the same impression held, but the strange sounds that he uttered soon showed that he was speaking a language entirely his own. He was asked to read an article from the newspaper, and he did so with a properly modulated voice as though he understood it, but what he said was quite incomprehensible. Reading it the second time he exactly reproduced the same sounds to express the various words.

Questioned upon what he had read, he convinced us that he quite understood the meaning of the article. Besides, we have had frequent opportunities of observing that he can enjoy an amusing book as well as any other child.

It would have been impossible to convey to the Society any idea of the language of the child as we first heard him, but for the phonograph ; and our ability to demonstrate by means of this instrument, the condition of his speech more than a year ago, is due to the courtesy of the Phonograph Company.

We have attempted in Table I to show the peculiar articular sounds, simple and combined, which he used to express the words and syllables of our language.

Table I.

(A) Alphabet as pronounced by Alfred P—. When not otherwise indicated the letters have their usual value.

a	b	c	d	e	f	g	h	i
ah	b	c	d	e	ahth	yee	ahsee	ah

j	k	l	m	n	o	p
eeyah	tah	ahl	ahm	ahdĭ	ū (as in frugality)	p

q	r	s	t	u	v	w
tū	ah	ahsee	t	u (as in few)	yee	dahbeyew

x	y	z
aht	eeyah	yahddĭ.

(B) The Lord's Prayer as pronounced by Alfred P—.

Our	Father	which	art	in	heaven,
Ah	Sahyee	yee	ahtee	ee	ahyah

hallowed	be	Thy	name.	Thy
hahdō (o as in hero)	bee	Dah	ah	Dah

kingdom	come.	Thy	will
tdō (o as in hero)	töm (o as in son)	Dah	yee

be	done	in	earth,	as	it	is
bee	dah	ee	ahtsee	ah	eet	tee

in	heaven.	Give	us	this	day	our
ee	ahyah	Yee	ahtsee	deesee	dah	ah

daily	bread.	Forgive	us	our
dahdĭ	bahdĭ	Dōsōyee (os as in hero)	ahsee	ahee

trespasses,	as	we	forgive	those
sahpeedee	ah	yee	dōsōyee (o's as in hero)	dō (hero)

who	trespass	against	us.
ū (as in frugality)	sahpee	ahdahthō (hero)	ahsee

Lead	us	not	into
Leedĭ	ahsee	out (ou as in loud)	eetō (hero)

temptation,	but	deliver	us	from
tahptahsĭ	baht	deeĕdĭ	ahsee	soum (ou as in soul)

evil:	For	Thine	is
eeew (ew as in Matthew)	Soud (ou as in soul)	Dahdĭ	ee

the	kingdom,	the	power,	and	the
dee	teedō (hero)	dee	pardĭ	ahd	dee
glory,		for	ever	and	
doudĭ (ou as in loud)		sō (hero)	ahyah	ahdĭ	
ever.	Amen.				
ahyah	Ahyah				

We will specially draw attention to the following points which have struck us during frequent interviews with this child.

(A) He always employs the same sounds or combination of sounds to express aloud any given syllable or word in our language, and however often he is asked to repeat it he always employs the same sound or combination of sounds. Inasmuch as this peculiarity constitutes the essential feature of this kind of case, and inasmuch as he thus always speaks a language of his own, we would venture to suggest the word "idioglossia" as a good name for this condition.

(B) His language is so bizarre that any detailed criticism with the idea of classifying the method by which he seems to be guided in his expression of words is beyond us, yet we observe besides the facts insisted on in the former paragraph he uses the word "sahpee" to express "trespass" and "sahpeedee" to express "trespasses," thus suggesting a primitive attempt at inflection. Also the initial "th" seems invariably to be transformed into "d," but this is a common defect in children.

(C) This idioglot language is in this lad almost musical. It flows easily from his lips, and, as will be seen by reference to the table, he makes frequent use of his liquid sounds (l, m, n, r) and his vowel sounds; for example, "heaven" is "ahyah," "hallowed" is "hahdō," and the closing words of the Lord's prayer also illustrate this fact.

(D) He does not appear to employ true gutturals, and this adds force to our last remark.

We have already mentioned that he writes English correctly to dictation, and this is most remarkable considering the hopeless jargon of his spoken language.

We have illustrated by the phonograph and described the condition of the child sixteen months ago, and although his speech has much improved the defect is still very evident.

The treatment that has been carried out under our suggestion has been not only careful and constant tuition in articulation, but isolation as far as possible from anyone who would not constantly correct his mistakes; he has also been separated from his brother and sister.

Robert J. P. P—, æt. 10½, was educated at the same schools as his brother from the age of five to nine, and his defect of speech has been present ever since he began to talk. He has always seemed an intelligent lad. He has acquired the same degree of education as his brother. He like his brother speaks a language of his own, but writes correctly to dictation.

It will be seen on reference to Table II that the peculiarity of his speech is less marked than in the case of his brother, and he uses the guttural letters. This lad can be to some extent understood when he speaks, and all we have said of his brother's condition applies to him, though in less degree. Physically he is as sound as his younger brother, but it is noticeable that he is left-handed, and in writing he is almost ambidextrous.

Table II.

(A) Alphabet as pronounced by Robert J. P. P—. Unless otherwise indicated the letters have their usual value.

a	b	c	d	e	f	g	h					
ahsee	b	c	gee	e	f	dzee	aisee (a as aye, i as eye)					
i			j			k	l	m	n	o	p	
aee (a as aye)			zaee (a as aye)			kee	l	m	n	o	p	
q	r	s	t	u				v		w		
q	ah	ees	kee	ū (u as in frugality)				wee		dahbeeyew		
x	y	z.										
ĕs	y	z										

(B) The Lord's Prayer as pronounced by Robert J. P. P—. The words are spelt in their usual manner when he pronounced them as ordinary persons do.

Our	Father	which	art	in	heaven,	hallowed
Ah	Fahwee	wĭs	aht	ee	eewee	hahdee

be	Thy	name.	Thy	kingdom
be	Di (i as eye)	nahĭm	Di (eye)	keedŏm (o as in got)

come.	Thy	will	be	done	in	earth, as
come	Di (eye)	wĕll	be	gahd	ee	ef ahsee

it	is	in	heaven.	Give	us	this
eet	ees	ee	eewee	Zwivĭ (first i as eye)	ahsee	dĭs

day	our	daily	bread.	Forgive
gaee (a as aye)	ahee	gayee (a as aye)	bĕd	Forgive

us	our	trespasses,	as	we	forgive
ahsee	ahee	sahpeesees	ahsee	we	forgive

those	who	trespass	against
dōsee (o as in hero)	who	sahpĭs	agaeesee (both a's as aye)

us.	Lead	us	not	into
ahsee.	Eedee	ahsee	not	eekew (ew as in Matthew)

temptation,		but	deliver	us	from evil:
keekasŏm (a as aye)		baht	geewee	ahsee	vŏm eewĭl

For	Thine	is	the	kingdom,	the
For	Dahee	is	dee	keedou (ou as in loud)	dee

power,	and	the	glory,	for	ever
pahdĭ	ahd	dee	gōri (or as in anchor)	for	eewee

and	ever.	Amen.
ahd	eewee	Ahmee

On making this boy say the elementary sounds, we obtained the following results: He said āī for ê, w for v, k for t, g for d, f for th, d for ð, n for ng; o was impure, l at the beginning of a word was dropped, but the final l was correct; r was quite incomprehensible, all double consonants were difficult; p, b, f, k, g, m, n, s, and z were all correct. He was very bad at combining sounds to form words.

The speech of the sister, Alice P—, æt. about 14 years, like that of many other children, is only slightly defective.

Her defects are, she employs v and f for "th," and drawls out some words, as "quarter," which she pronounces "keyoughtter." She is right-handed, but can write fairly well with the left hand. She is physically perfectly healthy.

All these children have been brought up together. They can understand one another after a fashion better than either of us can understand them. In a case quoted by Romanes, the reference to which we give later, a child aged 4½ years was the subject of idioglossia, but her younger brother was able to converse with her in her own idioglot language, although he also talked childish English.

We have endeavoured in the account of these idioglot children truthfully to record facts, and have added the conclusions which they seemed to warrant, but we feel far from being able to offer any satisfactory explanation of the infirmity from which these children suffer.

That it is congenital seems undoubted, it was noticed as soon as the children began to speak; this together with the fact that the worst case was the youngest in the family, makes it certain to our minds that it was not acquired by imitation.

It might seem to some that this is but a permanent form of domestic baby language; but a study of the tables will at once show that this is not the case, and also the vocabulary is too extensive. The resemblance of the language of these children in its form to English and the fact that they usually employ the proper number of syllables in each word, and further that they write correctly are arguments against the idea that idioglossia is an example of atavism in language.

In some of the few other cases which have been observed it has been noticed that the subjects of idioglossia are left-handed, or have their viscera transposed; it will be seen that the former defect was present in one of the children here described, but both defects may possibly be mere coincidences.

A child similarly affected was shown to the Neurological Society last year by Dr. W. B. Hadden. This patient was almost cured by careful training. Romanes ('Mental Evolution in Man,' p. 138) quotes two cases from American literature. He brings them forward to show that children, like animals, will occasionally invent arbitrary signs constituting a language of their own, but he, like us, is unable to give any satisfactory explanation of the cases he mentions.

Romanes' cases are not analysed in detail and are merely quoted by him to illustrate special points which he is discussing. We believe that Dr. Hadden's cases and those which we bring before the Fellows of this Society, this evening, are the first in which full accounts of the infirmity and its progress have been given in this country.[1]

[1] Since this paper was read other cases, under the title of "Formation of Language," have been recorded in 'Nature,' March 26th, 1891, p. 491, and April 9th, 1891, p. 534.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 95.)

A CASE OF DEFECTIVE ARTICULATION.

BY

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Received February 10th—Read March 10th, 1891.

Haislett S—, æt. 8½ years, was admitted under my care into the Evelina Hospital on September 14th, 1890, for imperfection in speaking, and I recognised it as being a similar case to one of Mr. Golding-Bird's cases, which I had previously seen with him.

The boy's father died of phthisis six years ago, and his mother is rather slight and pale, but otherwise healthy.

Mrs. S—'s sister's son was an idiot. He went to Earlswood at six years of age, and died there six weeks afterwards, probably from pulmonary mischief.

Haislett has one sister, ten years old, who is well-formed healthy-looking, and has dark brown hair and blue eyes. She is right-handed and talks quite naturally.

The boy had three fits when he was eight months old; measles at four years, and scarlet fever after the measles. He walked at ten or eleven months, and had his teeth at the usual time. With the exception of "dad-dad" and "mam-mam" he showed no signs of talking till about two years of age; and then began to make known his wishes by signs or by an imperfect speech, in which unintelligible words were used. The addition of the

syllables "ida" and "eeda" was noticed when he first began to talk.

On admission he was a ruddy, healthy-looking, well-formed boy with dark-brown hair and blue eyes, and intelligent face.

He presents no obvious physical defect, and is not left-handed. His organs are healthy. There is no fissure in the hard or soft palate, but the palate presents a rather high arch. He understands all that is said to him, but his own speech is almost unintelligible, though his sister, aged ten, can understand him to a certain extent.

His alphabet at this time was as follows :—

a	b	c	d	e	f	g
ādĕ	bēder	dēder	dēder	ēyer	arder	dgeeder

h	i	j	k	l	m	n	o
aidow	aider	diader	dader	aider	em	en	ōĕr

p	q	r	s	t	u	v
bēder	u	arder	aidow	deder	ūer	vēder

w	x	y	z
doubbe ouer	aidow	whyděr	diader

It was always repeated in a low voice indistinctly, and its peculiarities were the imperfect pronunciation of some consonants, and the addition of the syllables "āider" or "ĕr" to almost every one of the letters. The imperfection in the consonants will be seen to be largely in the failure of the hard consonants such as p, t, s, which require greater force of articulation than some others; thus, t is given as d, p as b, k, c, s, and z, are either given as d, or shirked altogether as consonants, and supplemented by the familiar addition "dĕr," and q was only a vowel sound.

The nurses and the residents attempted to improve his articulation by making him speak to dictation, and on September 25th it was reported that there was very great improvement in the child's speech. He could now say the alphabet fairly intelligibly, but still had great difficulty about k, and did not distinguish between p and b.

October 10th.—He was still improving, but was difficult to understand.

He was re-admitted on October 28th, having lost any improvement he had previously gained, and he left again on November 9th saying his alphabet fairly well.

Hearing that Mr. Golding-Bird and Dr. Hale White were going to bring their cases before this Society, I was reminded of Haislett S—, and got him to come to the hospital on December 20th, and as a record I requested him to say the Lord's Prayer to my dictation. It ran as follows:

Our	Father	which	art	in	heaven,
Ouarda	Fararda	id	arda	a	ha āida.
Hallowed	be	Thy	name.	Thy	kingdom
Howada	bedă	adāīda	dene	aidă	dēdă
come,	Thy	will	be	done	on earth as
dum	Thy	will	be	done	on eardă as
it is	in	heaven.	Give	us	this day our
id is	in	aidă	Dedă	ud	dud ou
daily	bread,	and	forgive	us	our trespasses
daily	bread	an	fudēdă		ou tredpadēdă
as	we	forgive	them	that	tresspass
ad	we	fordēda	um	dad	tredpada
against us,	and	lead	not	into	temptation,
ădа	and	eda	nod	eda	ida ada
but	deliver	us	from	evil.	For
ba	lēvĕr	a	evo-ulva		For
Thine is the kingdom,		the	power	and	the
di-de	dum	the	pour	an	de
glory,	for	ever	and	ever.	Amen.
dorēdă	for	edda	ad		Amen.

It was obvious on this occasion that he could with an effort speak better than this; at any rate, if a word were repeated to him several times, his later attempts were nearer and nearer to the correct pronunciation.

On February 3rd he came into the hospital again, and on February 4th I got the following from him :

Alphabet.

a	b	c	d	e	f	g	h
ā	bee	scedă	deedă	eeă	ef	dgēe	esst

i	j	k	l	m	n	o	p	q
ai	dā	dā	ell	emm	enn	ōh	bee[1]	tchu

r	s	t	u	v	w	x
arr	ess	dee[2]	yu	vee	dubber-yu	ess

y	z
wy	zed

The Lord's Prayer.

Our	Father	which	art	in	heaven,	hallowed
Ouer	Faerdĕ	we	ad	ā	evven	alloēedă

be	Thy	name.	Thy	kingdom	come,	Thy
bee	Thy	name	Thy	deedum	tum	Thy

will	be	done	on	earth	as	it	is	in
wĕe	be	done	on	earss	ad	it	is	in

heaven.	Give	us	this	day	our	daily
evven	Di	udde	sĕr	day	our	daerda

bread,	and	forgive	us	our	trespasses	as
bread	an	fordive	uddĕ	our	trepar eeda	adde

we	forgive	them	that	trespass	against	us,
wee	fordive	tem	deddĕ	trepuss	adu	ăāīdă

and	lead	us	not	into	temptation,
a	lead	ūddĕ	nod	intoo ă	temdā ă aidă

but	deliver	us	from	evil.	For	Thine
but	deliver	āidă	from	eevool	For	dine

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[1] Between p and b.

[2] Between t and d.

is	the	kingdom,	the	power	and	the
is	a	deedum	the	power	an	de

glory,	for	ever	and	ever.	Amen.
dorhēeă	for	evver	an	evver.	Amennă.

He has been to school; he can read little words, and can write in a child's round hand to dictation, and can copy words from a book.

Here, again, it was clear that he was amenable to instruction; often by repeating a sound he would try to imitate, and each later attempt would be more successful. It was chiefly when he repeated a string of words or a phrase after me that he became unintelligible, and the apparently involuntary affix "eeda" or "ida" was pronounced.

While I was sketching out this communication, Dr. Hadden kindly sent me a copy of his article in the 'Journal of Mental Science' for January, 1891, upon certain defects of articulation; and it is obvious that his two cases, the cases of Dr. White and Mr. Golding-Bird's, and my own, are practically the same form of defect, and to my mind they are certainly defects of articulation and not a new language. In my own case it seems so easy to recognise that most of the mispronunciation comes from a shirking of the more difficult sounds, especially the substitution of soft sounds for hard ones, or the omission of a consonant entirely in some cases. The improvement, also, with a very imperfectly organised attempt at treatment, bears in the same direction. When I first saw one of Mr. Golding-Bird's cases I was inclined to think that defective education was the cause of it almost entirely, and that there was no necessity to believe in a congenital defect. I am, however, prepared to admit that there is most likely some defect in the central articulating processes, which incapacitates the subject of this disorder for the perfect co-ordination or transmission of impulses to the articulating muscles. There is one feature in my case which is something more perhaps than defective

articulation and that is the addition of "ida" or "eeda" to so many words—a fault which was much more prominent a few months ago than now. It reminds me of a childish trick amongst school-boys, of trying to disguise their speech by adding some simple syllables to the end of each word spoken, by which the words are partly concealed in an almost continuous monotone. But even with this resemblance to a sham language, I think the affix in this case must be otherwise explained. It is not exactly a defect, because there is something added; and there are two ways in which it might come about.

The hearing of these patients is said to be normal; but it has occurred to me as not impossible that their auditory organs may interpret all simple sounds with a terminal buzzing or other sound, which their speech organs transmit as "ida" or "eeda." But I am not inclined to hold this view; the rapidity with which my patient has diminished this peculiarity is opposed to it. Another explanation is that the central organs have a defective control over the movements of the tongue, when once it has started on an order, and that thus this dental sound is produced.

As to naming, I hope we shall not be too hasty. I must confess I do not like "idioglossia." Firstly, because I would, if possible, restrict the word "γλῶσσα" in medicine and surgery to the organ, the tongue, and would not use it for language. Secondly, because I do not think this defect is a language in the more usual sense of the term, as for instance, the French language distinguished from the English. Thirdly, because I do not think the performance is peculiar in the sense used by Dr. White and Mr. Golding-Bird; that is, that the child always uses the same sound in his speech for the same words in ours. Dr. Hadden speaks of variations within certain limits, and I can say the same of my case, and yet I do not think any one can doubt the cases belong to the same group.

If, however, ἴδιος in the sense of peculiar or curious is

retained, I should be more inclined to speak of "idio-arthria," peculiar articulation—or "idiophasia," in the most general way, peculiar speech. I only throw these out as suggestions, and would willingly leave it to a committee to say what the name should be.

As to treatment, Dr. Hadden has no doubt rightly laid it down; and Dr. White and Mr. Golding-Bird speak in the same sense. What attempts I have made in this boy's case have been in the same direction, that is of careful oral instruction; but I have not seen my way to isolation in this instance. His temporary improvement has been so far good, and I am so far encouraged by hearing the results in Dr. Hadden's case, that I shall again make efforts to better his condition.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 95.)

ON THE TREATMENT

OF

STRANGULATED HERNIA

WHEN THE INTESTINE IS GANGRENOUS OR ULCERATED.

BY

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I HAVE twice had the misfortune to operate upon cases of strangulated hernia in which the intestine was gangrenous or ulcerated. The first case I treated in the usual way; the stricture was divided and the intestine laid open and fixed with sutures, some omentum being also removed. After living four days the patient died, without having had proper relief, although a finger could be pushed either up or down the intestine. No peritonitis was discovered, and the fatal ending seemed rather mysterious. In the second case a part of the bowel, which had been ulcerated by Gimbernat's ligament, gave way after it had been returned. I performed laparotomy by a median

incision, and made an artificial anus; the peritoneum was washed out and drained. The obstruction was completely relieved, but the patient died next day with the symptoms of septic intoxication.

Nothing could be less satisfactory than these results. The first case quite destroyed my confidence in the usual plan of treatment, because it failed even to relieve the obstruction. I surmised that the bowel did not empty itself either because it was paralysed, or because of the condition of the herniated part, which was obviously swollen and not freely pervious. I therefore determined to try and ascertain from the surgical records of St. Bartholomew's Hospital what might be the result of treating the gangrenous intestine by free incision. Moreover, I was aware that information of this kind was urgently needed to enable this method of treatment to be compared with others.[1]

Careful records of the surgical practice of St. Bartholomew's Hospital have been kept for the last seventeen years, and I am deeply indebted to the surgeons, under whose care they were, for the kind and cordial way in which they have allowed me to make use of the cases, and to Mr. Bowlby for his assistance in helping me to obtain the notes. I have found thirty-five cases of hernia[2] in which the strangulated gut was ulcerated or gangrenous, and which had all been treated in the manner recommended so strongly by Sir William Lawrence,[3] namely, by "a free incision through the mortified part, in order to unload the distended intestinal canal; or, if the gut should have already given way, to divide freely the integument and sac, and to leave the subsequent process of cure entirely to nature." The stricture was also to be

[1] See concluding paragraph of Mr. Makin's paper on a "Case of Artificial Anus treated by Resection of the Small Intestine," 'St. Thomas's Hospital Reports,' vol. xiii.

[2] These thirty-five cases were made up as follows:—Men 13, women 22. Femoral herniæ 23, inguinal herniæ 10, umbilical herniæ 1, and ventral herniæ 1. See also Postscript for five additional fatal cases.

[3] 'A Treatise on Ruptures,' 5th ed., 1868, p. 364.

divided, if necessary, for the proper flow of fæces.[1] This advice is supported with such a glowing account of the marked benefit experienced by the patient, the casting off of sloughs, the restoration of the continuity of the bowel, and the closure of the wound, that it is easy to understand its general acceptance. But such a sanguine view is by no means warranted by the results of the operation when tested by a series of cases taken without any selection. Putting aside the two fatal cases which were under my own care and which have already been mentioned, out of the thirty-five collected from the St. Bartholomew's records, thirty-one died (88·57 per cent.). The four survivors went through a long and tedious convalescence and submitted to repeated operations for the cure of fæcal fistulæ.

A brief epitome of their history may give an idea of the nature of these cases which, it is no exaggeration to say, were very extraordinary. For instance, at the end of 1869, a woman, aged thirty-nine years, and who was eight months pregnant, came under the care of Sir James Paget, with a left inguinal hernia which had been strangulated twenty-four hours. Twelve hours later the sac was opened; the intestine was gangrenous and was incised and stitched to the edges of the wound; the stricture, which was very tight, was freely divided. For the first twenty-four hours there was no relief; on the third day she was delivered of a living child. During the confinement intestines protruded through the wound, but were put back again, and in less than three months the wound was quite healed, and she was restored to perfect health.

Again, in a second case, a woman, aged fifty-three, was operated upon by Sir William Savory in 1884 for a strangulated femoral hernia of the right side. The intestine was gangrenous and was incised, but the stricture was not divided. The woman survived and a spur formed, for which the enterotome was twice used, and

[1] Loc. cit., p. 342.

afterwards a plastic operation was performed, and a year later the fistula had healed.

In the third case, the patient was a man, fifty-four years of age, who had had a strangulated inguinal hernia for some days but who was too collapsed to give any history. The intestine was gangrenous and was laid freely open, and the stricture dilated by the finger pushed into the interior of the intestine. The patient recovered from the operation, but eight months later there was still a fæcal fistula. I have been unable to learn whether this ever closed.

The last case was almost as extraordinary as the first. The patient was a woman, aged forty-four, who was under the care of Mr. Willett. In October, 1882, she came into hospital with a ventral hernia which had been strangulated four days. It contained two feet of gangrenous intestine, which was laid freely open with prompt relief. After the dead tissues had separated she progressed favourably, although her nutrition was defective, and at the site of the hernia two apertures were left; a large one which belonged to the upper end of the bowel, probably the upper part of the jejunum, and which discharged ill-digested food; and a smaller one which was discovered with difficulty, and was supposed to belong to the lower end of the bowel. An enterotome was applied (February 23, 1883) to the tissues between these openings, but without effect, and, therefore, an exploratory operation was performed (April 24th, 1883). The peritoneal cavity was opened, but only the upper end of the bowel could be found, and was left *in situ*, and the colon, which lay close at hand, was fastened in close proximity to it, with the view of their subsequent union. This was afterwards done (May 31st, 1883) by an operation by which the peritoneum was again opened. As this attempt did not succeed, yet another operation was done; the peritoneum was opened for the third time (September 26th, 1883) and the ileum and colon again sutured and covered with a flap of skin. She left the hospital on December

13th, 1883, with a small fæcal fistula, which afterwards quite closed. Mr. Willett (to whom I am deeply indebted for the kind and cordial way in which he explained the case to me) saw her a year after the operation, quite recovered.

I do not propose at present to comment upon these recoveries. They show very clearly the difficulties and dangers which await the small minority who survive the original operation.

We will, therefore, proceed with the analysis of the thirty-one fatal cases. In these the end was seldom long deferred. The brief abstracts of them, which are given at the end of this communication, show that no less than twelve died the day of the operation; five upon the next day; four on the third; one on the fourth; two on the fifth; two on the sixth; and one each on the seventh, eighth, tenth, fifteenth and thirty-sixth days respectively. The great mortality which followed closely upon the operation is easily accounted for by the desperate condition of many of the patients at the time of their admission, but after these have been deducted, a fair proportion is left of those who had sufficient strength to rally and survive for some time.

It may be remembered that there was no relief in the case in which I myself performed the usual operation of opening the gangrenous intestine. Nothing came from the bowel, although a finger could be thrust into its lumen in either direction. Let us turn to the records to see whether this want of relief is an unusual circumstance. It is obvious that the cases which died very soon after the operation can throw very little light upon the point. But in fourteen cases which lived beyond the third day there seems to have been no fæcal relief in two, and very imperfect or delayed relief in three others. In the case of a woman, operated upon by Mr. Willett (Case 11), it was ascertained that the gangrenous loop was still compressed by some omentum, which had been left in the ring; so that the bowel would not even transmit air. At the same

time the intestine above the obstruction was distended, the rest being empty. In a case (Case 9), operated upon by Sir William Savory, there was never any free discharge from the wound, and the patient died on the fifth day. It was afterwards found that the stricture had been freely divided, and a loop of jejunum stitched in the wound. The intestines above this loop were distended, whilst the rest were empty. There was no fæcal extravasation or peritonitis, and the opening into the bowel was quite free.

The notes of all the cases have been made with no particular object in view, yet it is the rule, rather than the exception, to find it stated that the intestines above the opening in the gangrenous gut were distended whilst the parts below were empty. The emptiness of the lower part is what we would expect, but the distension of the upper part is, to say the least, highly significant.

In another case it is said that there was no relief until the sixth day after the operation. It was then too late, for the patient soon died, exhausted by sickness (Case 6). In the case of a young man (Case 5), operated upon by Mr. Morrant Baker, no flatus passed from the wound for many hours, and nothing else until the fifth day; the patient dying on the seventh. And in another case under the same surgeon (Case 4), the relief seems from the notes to have been unsatisfactory. Another case operated upon by Mr. Langton (Case 19), the patient died on the second day without having had relief. There was, however, a slight general peritonitis, but no fæcal extravasation.

The impression left by these cases is, that the incision into the gangrenous intestine cannot be relied upon to relieve the fæcal obstruction; or that the relief may come too late to save the patient's life.

Sometimes the presence of peritonitis may account for the want of relief. This may have been so in the last-mentioned case of Mr. Langton's; or in that of a woman upon whom Mr. Willett operated (Case 18). This patient died the day after the operation, without relief, and with

vomiting and collapse, and it was ascertained that there was general peritonitis due to fæcal extravasation.

But in the other cases in which the obstruction continued after the intestine had been laid open, there was no peritonitis, and the absence of relief was due either to paralysis of the intestines or to the morbid condition of their damaged portion. Either of these may suffice to prevent relief quite apart from anything connected with the gangrenous loop having been left within the hernial orifice. If evidence be wanted to show that the damage done to the intestine is alone sufficient to prevent the passage of fæces through it, let us glance for a moment at the accounts of the post-mortem examination of cases of strangulated hernia which died unrelieved after the intestine had been returned into the abdomen. In a case of Mr. Willett's, which died upon the second day, it is said that a small portion of ileum was found very dark and congested, and did not look as if anything had passed through it since the strangulation ; the gut above being dilated and that below constricted. It is also remarked that there was no peritonitis. In another case the knuckle of ileum, which had been strangulated, had ulcerated through, but fæcal extravasation had been prevented by an adhesion between it and the mesentery. "Above the place the gut was more distended than below, and it did not seem that anything had passed the seat of stricture since the operation." In a case operated upon by Sir William Savory, and which died without the obstruction or the sickness having been relieved, a small knuckle of intestine lay loosely in the femoral ring (which had been freely divided) and the intestine above was distended, but below it was empty.

It would be easy to multiply instances of this kind, but those which have just been given may suffice to show that, as most surgeons are already aware, the condition of the intestine itself, quite apart from any position which a loop of it may occupy, is quite enough to prolong the obstruction.

Without doubt the absence of relief is due in some

cases to the paralysis which follows the distension, and violent peristalsis. The late Dr. Brinton[1] laid stress upon this point and Mr. Treves[2] has more recently referred to it. But we have also to note that the morbid condition of the injured bowel may offer a mechanical obstacle to the onward passage of the fæces. In the case under my own care the swelling and œdema of the mucous membrane of the incised intestine was very obvious, and may have prevented the escape of the contents. In another case, in which the bowel was returned, it is said "that the congested gut felt quite solid, and on cutting it open the walls were found much thickened, and with scarcely any contents." And in another, that the loop which had been strangulated was "black and thickened." It seems reasonable to suppose that such conditions as these must offer a mechanical obstacle to the onward propulsion of the intestinal contents.

But besides being almost occluded by swelling of its coats, the injured loop of bowel may be in such a condition as to prevent the passage of fæces by stopping the onward progress of the peristaltic wave. Dr. Brinton[3] has proved this quite clearly, and museum specimens and the post-mortem notes show that it is not unusual for the muscular and mucous coats of the intestine to have been divided by the stricture. Also, in addition, the mucous membrane may be inflamed, covered with lymph, ulcerated, sloughing, or, as in cases such as those which we are considering, in a state of gangrene.

In addition to the probability of its not relieving the obstruction the operation of opening the gangrenous bowel leaves the patient exposed to other dangers. The spread of the gangrene or of the ulceration is one of these dangers. A patient of Mr. Morrant Baker's (Case 4) died of collapse on the eighth day, and it was ascertained that there was diffuse peritonitis and fæcal extravasation

[1] 'Intestinal Obstruction,' by William Brinton, London, 1867, p. 4.

[2] 'Intestinal Obstruction,' London, 1884, p. 401.

[3] Loc. cit., p. 4.

due to the intestine having ulcerated and given way at the seat of stricture; the notes also state that the gangrene had extended from the intestine, which had been left in the sac, to that within the abdomen. In another case under the care of Sir William Savory (Case 2) it is said that gangrenous ulceration had spread as in the last case, and permitted fæcal extravasation, with subsequent peritonitis. In another case, which died on the third day, the gangrene seems also to have spread, for it is stated (Case 14) that there was extensive sloughing of the small intestine; more than could be explained by the strangulation. Again, in a case of Mr. Willett's (Case 18) fæcal extravasation had been permitted by an ulcerated aperture in the constricted part of the intestine. In another case operated upon by Mr. Howard Marsh (Case 24) the gangrene did not spread, but "the intestine above the hernia was much distended and its peritoneum was ruptured in places and almost gangrenous." Lastly, and this is perhaps a less grave occurrence, the gangrene may spread to the edges of the wound, as it did in a case of Mr. Langton's (No. 19). Thus, I think, we may safely assert that the operation of incising the gut affords no security whatever against the local extension of the gangrene or ulceration, nor is it certain to relieve the distension which may be causing them elsewhere.

After the gangrenous intestine has been incised and left in the sac there also remains the danger of septic inflammation spreading backwards to the rest of the peritoneum. There was general peritonitis in fourteen of the thirty-one fatal cases given at the end of this communication; in seven others it is specifically stated that there was none; and in the remainder the subject is not mentioned. Although it is in accordance with our experience to find a focus of inflammation near the gangrenous bowel in these case of general peritonitis, it is seldom referred to in the post-mortem notes. But in the case of a very old woman, who lived four days, it is stated that a focus of peritonitis was found in the vicinity of the opened and

gangrenous bowel; and that there was injection of the rest of the intestines, without extravasation of fæces (Case 10). In another instance the peritonitis seems to have been associated with, and perhaps caused by, extravasation of fæces from the hernial sac into the cellular tissue of the right iliac fossa outside the peritoneum (Case 15).

In a fair proportion of cases, however, there seems to have been no trace of peritonitis[1], and we may remark in passing, that this affection may sometimes be diagnosed when it does not exist, as, for instance, in one of the St. Bartholomew's cases (Case 17).

The fatal cases also show that sub-peritoneal fæcal extravasation must be reckoned as one of the dangers of incising the gangrenous intestine (Case 15).

Should the patient survive any length of time after the gangrenous bowel has been laid open, a danger of quite a different kind has to be encountered, and nutrition may prove a source of anxiety. In a case of the late Mr. Callender's (Case 1), which lived thirty-six days after the bowel had been opened, although plenty of food was taken and nutrient enemata administered, the patient did not thrive, and finally died of exhaustion. Perhaps the jejunum had been opened, but unfortunately a post-mortem was not allowed. In another case, which died on the fifteenth day (Case 2), the patient suffered from inanition and her food passed from the wound little changed, but on this occasion it was ascertained that a loop of ileum, two and a half feet from the ileo-cæcal valve, had been stitched to the wound. But in another case which died on the fifth day, the lower end of the jejunum had actually been stitched in the wound (Case 9); and in yet another, the beginning of the ileum had been opened (Case 27). Therefore it is evident that although, as is well-known and accepted, the lower end of the ileum is most commonly engaged in hernial sacs, yet we may have unwittingly to deal with a part very much nearer

[1] Mr. Bryant says that peritonitis exists in about 69 per cent. of fatal cases 'On Hernia with an Analysis of 126 Fatal Cases,' pp. 12 and 14.

the stomach. Sir William Lawrence[1] gives a number of instances of the same accident, and in which the patients died of inanition, and many other surgeons have done the same, so that the danger is evidently not one which can be ignored.

It follows from the above that should the surgeon elect to carry out the usual treatment and incise the gangrenous bowel, he leaves his patient exposed to the following imminent perils: 1. Non-relief of the intestinal obstruction. 2. Spread of the gangrene or ulceration, and fæcal extravasation (intra- and extra-peritoneal). 3. Spread of septic peritonitis from the hernial sac. 4. Death from inanition.

Instead of incising the gangrenous intestine, the following alternatives have been adopted: The gangrenous bowel has been removed at once and the ends of the healthy bowel united—immediate suture. Next, the gangrenous bowel has been removed and an artificial anus established, with a view of uniting the ends of the intestine by a subsequent operation—secondary suture. Or, after the artificial anus has been established, it has been cured and the continuity of the canal restored by destroying the spur with Dupuytren's enterotome, or with an electrolytic clamp. I have also to add that Mr. Bennett[2] has recently recommended that the damaged bowel be returned within the abdomen after free division of the stricture; also that a large-sized drainage-tube be introduced into the ring to provide for the impending fæcal extravasation. This plan seems to promise better than the usual one of leaving the damaged intestine in the sac, but it is clear that it leaves a great deal to chance.

Dr. W. Körte's[3] recent exhaustive review of what he calls "the old and new methods of treating the gangrenous

[1] Loc. cit., p. 349.

[2] "Some Points relating to the Management of the Damaged Bowel in Strangulated Hernia,' William H. Bennett, 'Lancet,' October 18th, 1890, p. 806.

[3] 'Deutsche Medicinische Wochenschrift,' 1888, p. 829, No. 41.

intestine," affords us an idea of the relative mortality of the various methods.

It is, of course, difficult to ascertain the exact mortality of the usual plan of treatment, by incising the gangrenous bowel, but it is right to say that the mortality of the Bartholomew's cases is higher than that given by some surgeons, Reichel's[1] statistics giving a mortality of 52 per cent. for all plans of treatment, and Hænel's[2] a mortality of 54·2 per cent. Kocher,[3] on the other hand, lost seven cases, one after the other, and says that he has never seen one which has not eventually died on account of something connected with the artificial anus.

As regards the enterotome, we note that Körte, in his most recent writings, gives a list of 111 cases of anus prenaturalis treated with the enterotome, and of these only 11 died. But it is to be remembered that those who submitted themselves to this treatment were the survivors out of a very large number of cases of gangrenous bowel. Many of the patients, however, were not cured, because it is said that in 30 a fæcal fistula remained.

According to Schmidt,[4] the mortality after primary resection and sutures is 71 per cent., and after incision and artificial anus, 85·5 per cent., almost the same as in the St. Bartholomew's cases. McCosh, at the end of the excellent essay in which he records a successful case of primary resection and suture,[5] tabulates 113 cases of the same kind, of which half were successful.[6] Mr. Makins tabulates 55 cases with 29 deaths, a percentage of 52·7. Ferdinald Klaussner had 7 recoveries in 14 cases.[7]

[1] 'Deutsche Zeitschrift für Chirurgie,' Bd. xix, Heft 3, p. 230.

[2] Von Langenbeck's 'Arch.,' Bd. xxxvi, Heft 2.

[3] 'Correspondenzblatter für Schweitzer Aertze,' March 1st, 1886.

[4] Quoted by Körte.

[5] 'New York Medical Journal,' 1889, p. 285.

[6] A successful case is also described by Surgeon-Major W. R. Browne in the 'Indian Medical Gazette,' March, 1890, p. 89, *et seq.* Another by Mitchell Banks, 'Med. Soc. Proc.,' vol. viii, p. 279; also, K. A. Walter, 'Hygeia,' vol. xlviii, p. 263.

[7] 'Münchener, med. Wochenschrift,' Nos. 5 and 6, 1889.

The mortality of secondary sutures is less than after primary resection and suture. Reichel puts it at 37·8 per cent., Hertzberg says recent experiences have brought it down to 27 per cent.; Makins puts it at 28·4 per cent.; McCosh[1] collected 120 cases with a mortality of 52 per cent. However, we must bear in mind that those upon whom secondary suture is performed are the rare and infrequent survivors of a most fatal class of cases. Indeed, it is almost to be wondered that so many could have been collected, but in the tables of secondary suture for fæcal fistula cases due to a variety of causes are all grouped together. For instance, in Mr. Makins' tables the causes of the fistulæ were gangrene after hernia, gunshot wounds, blows, and wounds of the intestine, and, besides, the same cases are used over and over again by the different authors.

Thus it appears that as regards mortality the plan of incising the gangrenous intestine is at present the most fatal procedure to adopt; primary resection and suture comes next; then secondary suture; and, finally, least fatal of all, the application of the enterotome. However, it may be again said that before either of the last two methods can be put into practice, the patient must have had the exceptional good fortune to escape from a number of most fatal accidents.

The removal of the gangrenous bowel, and subsequent suture as usually performed, takes a considerable time, and is attended with shock, and therefore is often debarred by the condition of the patient. But it is a question how far we ought to be deterred. Hagemann[2] operated successfully upon an ill-nourished boy of twelve years of age, who before the operation began had cold extremities, a scarcely perceptible and rapid pulse, and commencing collapse. After an operation, which lasted

[1] Loc. cit., p. 285.

[2] "Casuisticher Beitrag zur primären Darmresection und primären Darmnaht bei gangränösen Hernien," R. Hagemann, 'Deutsche medicinische Wochenschrift,' 1889, No. 31, p. 627. Another successful case is mentioned in this paper, p. 629.

an hour and a half, this boy was revived with warmth and subcutaneous injections of ether, and afterwards made a complete and rapid recovery.

I venture to submit that the arguments and statistics which have been brought forward are in favour of performing primary resection in cases of hernia in which the bowel is gangrenous or ulcerated. Without doubt a distinction ought to be made between the two classes of cases. Where there is ulceration the destruction is sometimes circumscribed, and may only involve a part of the circumference of the bowel.[1] In most of these the resection of a very small portion of bowel is required, whilst in some it may prove sufficient merely to invert and suture the margins of the aperture, and it is possible to accomplish this through the original wound. The mesentery is never so short as to prevent a sufficient extrusion of the bowel.[2]

When the intestine is gangrenous a more extensive procedure is required. I have only been called upon to perform resection of the intestine upon one occasion, and that was for intussusception. I have never as yet done it in a case of gangrene following strangulation, and, therefore, I do not propose to discuss the operation at length. There are, however, some obvious considerations which may be mentioned. As soon as it has been decided that the operation ought to be performed, the patient should be carefully surrounded with blankets and hot water bottles, and stimulants for hypodermic administration prepared. The resection should be done through the original wound, which is to be enlarged without disturbing the gangrenous bowel, beyond what may be necessary for its thorough disinfection with perchloride lotion (1 in 5000) discharged from an irrigator.

In doing this the operator learns the extent of the gan-

[1] In this class of cases the strangulation is not infrequently of recent date, whilst in the other it is often of long standing and due to neglect, the patient being much exhausted.

[2] 'Hunterian Lectures on Hernia,' by C. B. Lockwood, 1889, pp. 35 and 51, &c.

grene, the presence or absence of septic peritonitis, or of fæcal extravasation. When all this has been ascertained, the peritoneum ought to be packed with sponges, and both ends of the bowel properly controlled—a clip made out of a flexible pessary is convenient for this purpose—and the gangrenous portion of the bowel removed, together with any gangrenous mesentery or omentum. A margin of more than half an inch ought to be left between the healthy and diseased tissues, and the collapsed distal bowel should be divided first, as the gangrenous portion and the distended end may then be drawn from the wound, and used as a spout to carry off the fæcal accumulation. This unloading of the distended bowel greatly facilitates the operation, and has an influence upon the subsequent result. The removal of a wedge of mesentery is contra-indicated. Although desirable, perhaps, on theoretical grounds, it leads to so much hæmorrhage and loss of time that the advantages are outweighed by the disadvantages. The actual enterorrhaphy may be done by Czerny-Lembert's method,[1] but it is possible that experience may show that Senn's[2] modification of Jobert's method may give the same favourable results in man as it has in animals. The plan is as follows : " The upper end of the bowel which is to become the intussusceptum is lined with a soft, pliable, rubber ring, made of a rubber band transformed into a ring by fastening the ends together with catgut sutures. This ring must be the length of the intussusceptum, from one third to half an inch ; the lower margin is stitched by a continuous catgut suture to the lower end of the bowel, which effectually prevents the

[1] Successes have been obtained by the operation of intestinal anastomosis by means of Senn's plates. This method seems to have been practised by Helferich, and is sometimes called " Helferich's operation," see Helferich, " Ueber die Ausführung der Herniotomie bei der Gangrän Verdächtigem Darm.," ' Cent. für Chirurgie,' vol. xvii, p. 56 (Kongress). Also " Ueber die Helferich'sche Operation bei der Gangrän Verdächtigen Hernien," L. Kredel, ' Cent. für Chir.,' vol. xvii, 1890. Also Arbuthnot Lane ' Clin. Soc. Trans.,' 1891.

[2] ' Intestinal Surgery,' Chicago, 1889, p. 168, *et seq.*

bulging of the mucous membrane, a condition which is always difficult to overcome in circular suturing. After the ring is fastened in its place, the end of the bowel presents a tapering appearance which materially facilitates the process of invagination. Two well prepared, fine juniper catgut sutures are threaded each with two needles. The needles are passed from within outwards, transfixing the upper portion of the rubber ring and the entire thickness of the wall of the bowel, and always equidistant from each other, the first suture being passed in such a manner that each needle is brought out a short distance from the mesenteric attachment, and the second suture on the opposite convex side of the bowel. During this time an assistant keeps the opposite end of the bowel compressed, to prevent contraction and bulging of the mucous membrane. The needles are next passed through the peritoneal, muscular, and connective-tissue coats at corresponding points about one third of an inch from the margins of the opposite end of the bowel, and when all the needles have been passed, an assistant makes equal traction on the four strings, and the operator assists the invagination by turning in the margins of the lower end evenly with a director, and by gently pushing the rubber ring completely into the intussuscipiens. The invagination accurately made, the two catgut sutures are tied only with sufficient firmness to prevent disinvagination, should violent peristalsis follow the operation." I am not aware that this method has been practised on human beings, but it seems worthy of a trial. After the bowel has been sutured and disinfected and dusted with iodoform, it is to be placed within the abdomen, and the hernial sac removed, and the ring closed in the usual way.

The after-treatment of these cases need not be discussed, and is to be carried out in accordance with established principles, except that the administration of opium is to be earnestly deprecated as conducing to further, and, perhaps, fatal, intestinal paralysis.

Postscript.—Since the foregoing was written I have been able, with the kind help of my friend Mr. Macgregor, to add the notes of the cases of strangulated hernia which were operated upon in St. Bartholomew's Hospital during the year 1890, and in which the intestine was gangrenous or ulcerated. They were five in number, which is rather beyond the average, and were all treated in the usual way, and all died. If we add them to the other 35 cases they bring the death rate up to 90 per cent.; but this statement needs qualifying because one of the patients (Case 36) died as the operation was being completed. The others survived, three weeks, three days, thirty hours, and nine hours, respectively. They bear out the conclusions already arrived at, especially as regards the failure of the operation of incising the intestine to give relief. A woman aged sixty-five (under Mr. Willett) lived for three days after the operation. During that time there was no fæcal flow from the wound, and the vomiting continued. At the examination, the intestine above the hernia was found greatly distended and inflamed, below it was flaccid and empty (Case 33). The enterocele was partial (Richter's hernia), and the notes say that after the operation flatus was passed per anum and the bowels acted slightly. A very old woman (Case 35) was also operated on by Mr. Willett, and although she lived nine hours, there was no fæcal flow from the wound, and it was afterwards ascertained that the intestines above the gangrenous part were enormously distended with fæculent fluid. It is also said that the sac was thickened, and seemed gangrenous.

The third case was that of a woman æt. 54 (Case 34), who was operated upon by Mr. Long (Sir William Savory's House Surgeon) for a strangulated femoral hernia. The bowel proved to be ulcerated opposite Gimbernat's ligament, and it gave way after having been reduced. It was therefore drawn down, laid open, and stitched to the edges of the wound. The patient lived

for thirty hours, but had during that time no relief by the wound. She is said to have died from shock, but at the examination they found general purulent peritonitis, together with some fæcal extravasation.

The last case I have to mention (Case 32) was that of an infant, æt. 5 weeks, who was under the care of Mr. Langton. The rupture was a congenital inguinal hernia of the right side, containing a loop of gangrenous intestine. The latter was freely opened, and the constriction dilated with forceps. The infant did well for a few days, its bowels having relieved themselves by the wound, but it died at the end of three weeks of general peritonitis, without effusion of fluid, but with much lymph and many adhesions. It is said that the intestines above the artificial opening were distended.

Thus the cases for the year 1890 bear out the statement that the usual operation of incising gangrenous or ulcerated intestine, in cases of strangulated hernia, cannot be relied upon to give relief. They also show the dreadful mortality of that condition when treated in the usual way.

Abstracts of Fatal Cases.

Case 1.—*Gangrene of intestine; death from exhaustion on the thirty-sixth day.*—In 1875 a woman, æt. 50, was under Mr. Callender's care, having had a right femoral hernia strangulated for two days. The sac contained inflamed omentum and gangrenous intestine. The stricture was divided and the omentum cut off; some of the intestine was returned, the rest opened and left in the wound. Although the patient afterwards took plenty of food, and had nutrient enema, she did not thrive, and died of exhaustion on the thirty-sixth day. No post-mortem examination was allowed.

Case 2.—*Gangrene of intestine; spread of gangrene; fæcal extravasation; general peritonitis; inanition; death*

on fifteenth day.—A woman, æt. 55, had had a right femoral hernia strangulated three days. The intestine was gangrenous, and Sir William Savory laid it open, and stitched its edges to those of the wound; the stricture was slightly divided. The patient suffered from inanition, her food passing out again from the wound little changed. She died on the fifteenth day, when it was ascertained that a part of the circumference of the ileum, two and a half feet from the ileo-cæcal valve, was engaged in the femoral ring, and was in a state of gangrenous ulceration, which, by its spreading, had permitted fæcal extravasation with subsequent general peritonitis.

Case 3.—*Gangrene of intestine; death on tenth day from exhaustion.*—In 1873 Mr. Morrant Baker had under his care a woman, æt. 56, who had had a right femoral hernia strangulated for eight days. The sac contained omentum and intestine, the latter gangrenous and collapsed. The stricture was freely divided, and two days later there was an abundant flow of fæces from the wound. The patient died of exhaustion on the tenth day, and it was ascertained that there was no peritonitis or obvious cause for her death.

Case 4.—*Gangrene of intestine; spread of gangrene; fæcal extravasation; death on eighth day.*—Last year Mr. Morrant Baker operated upon a man, æt. 58, who had reducible inguinal hernia on both right and left sides, and a strangulated femoral hernia on the right side of four or five days' standing. The latter contained gangrenous intestine, which was laid open without dividing the stricture or disturbing the adhesions about the neck of the sac. There was sickness on the second and third days after the operation, and relief seems to have been very imperfect. He died suddenly of collapse on the eighth day, the abdomen being much distended. It was ascertained that there was general peritonitis with extravasation of fæces into the peritoneum, due to the intestine having ulcerated and

given way at the seat of stricture. The notes also say that the gangrene had extended from the intestine within the hernial sac to that within the abdomen.

Case 5.—*Gangrene of intestine; death on seventh day; tardy relief.*—A strong young man, æt. 21, had had a right congenital inguinal hernia strangulated for three days. The intestine was gangrenous and perforated, and Mr. Morrant Baker opened it and stitched its edges to those of the wound. The stricture was not divided. No flatus passed from the wound for some hours, and no fæces until the fifth day, and he died on the seventh. It was ascertained that the intestines above the hernia were distended with flatus, and red and hyperæmic from commencing inflammation, but there was no lymph or fluid. Only a part of the lumen of the intestine was engaged in the stricture, and not more than half an inch of its length.

Case 6.—*Gangrene of intestine; tardy relief; death on the sixth day.*—A man, æt. 53, had had a right femoral hernia strangulated for twenty-four hours, but, owing to his refusal, no operation was done until twenty-four hours later. The stricture was divided and the intestine, which was gangrenous and ruptured, was stitched to the edges of the wound. The sickness continued, and there seems to have been no fæcal discharge until the sixth day, but the patient sank exhausted a few hours after. There was no fæcal extravasation or peritonitis, but the note says "that the intestine was completely cut through in its whole circumference, so that it would have been difficult to restore the continuity of the canal at any future period."

Case 7.—*Gangrene of intestine; death on sixth day after operation; general peritonitis.*—A man, æt. 41, was kicked by a horse in the right groin, four days after an old-standing right inguinal hernia became strangulated. At the operation the hernial sac was full of fæces and a longitudinal cut was made into the gut, and it was stitched to the edges

of the wound. The patient died six days later, having continued to vomit, and it was ascertained that the sutured gut was ileum and that there was general peritonitis.

Case 8.—*Gangrenous intestine; death on fifth day.*—A woman, æt. 57, who had had a right femoral hernia strangulated three days, was operated upon by Mr. Thomas Smith. The intestine was gangrenous and was laid freely open and the stricture divided. There was fæcal vomiting on the next day, and the obstruction seems to have been partially relieved by the operation, but the patient died on the fifth day after. At the examination it was ascertained that seven inches of gangrenous ileum, three feet from the ileo-cæcal valve, lay in the sac with recent adhesions about femoral ring. There was no fæcal extravasation or peritonitis, but the intestines were distended and swollen above the part which had been opened.

Case 9.—*Gangrene of intestine; death on fifth day; imperfect relief.*—A woman, æt. 64, had suffered from strangulation of a right inguinal hernia for four days. When Sir William Savory opened the sac it was full of fæcal fluid, and the intestine was collapsed. The intestine was laid open and stitched to the wound, and the stricture was freely divided. There was never any free discharge from wound or relief *per anum*, and the patient died exhausted on the fifth day. No fæcal extravasation or peritonitis were discovered, and it was found that the lower part of the jejunum had been stitched in the wound. Above this point the intestines were distended, below they were empty.

Case 10.—*Gangrene of intestine; death on the fourth day.*—A woman, æt. 72, had had a left femoral hernia strangulated for some days. At the operation a line of grey slough was found in the bowel at the seat of stricture, and therefore it was laid open and stitched to the wound.

There was a great flow of fæces from wound, but on the fourth day she died with much abdominal distension. There was no fæcal extravasation, but local peritonitis with injection of the rest of the peritoneum.

Case 11.—*Ulceration of intestine with gangrene of omentum; death on third day unrelieved by the operation; commencing peritonitis.*—Mr. Willett operated upon a woman, æt. 52, who had had a left femoral hernia strangulated for six days. The sac contained gangrenous omentum and small intestine, which latter was ulcerated through at Gimbernat's ligament. The gangrenous omentum was cut off, and the intestine united to edge of wound. The operation gave no relief and it was ascertained that it was still compressed by the adherent omentum, not even transmitting air. There was commencing peritonitis.

Case 12.—*Gangrenous intestine; death on third day.*—In 1878 Mr. Callender operated upon a woman, of uncertain age, who had had a femoral hernia of the left side strangulated for a week. The sac contained omentum, which was cut off, and intestine which was gangrenous and was left, the stricture having been divided. There was relief by the wound, but the patient died two days after the operation.

Case 13.—*Gangrene of intestine; death third day.*—A man, æt. 32, had had a right inguinal hernia strangulated for twenty-four hours. The operation was done by Mr. Willett, and the sac contained omentum, which was removed and the pedicle returned into the abdomen, and also intestine which was gangrenous, and was laid open and stitched to the edges of the wound, healthy intestine having been pulled down for the purpose. The patient died of weakness two days later, the bowels having been relieved. The examination showed no cause for the fatal ending.

Case 14.—*Gangrenous intestine ; death on third day.*—A woman, æt. 44, who had had an umbilical hernia strangulated four days, was operated upon by Mr. Thomas Smith. There was extensive gangrene of the intestine which was laid open, the stricture having been previously divided. The patient died on the third day, and it was ascertained that there was fæcal extravasation into the peritoneal cavity. There was extensive sloughing of the small intestine, and this was greater than could be explained by the extent of the strangulation.

Case 15.—*Ulceration of gut ; death the day after operation; extra-peritoneal fæcal extravasation.*—A man, æt. 34, was operated upon by Mr. Morrant Baker for a femoral hernia of the right side, which had been strangulated some days. The skin over the hernia was œdematous and inflamed, and there was a gush of fæces as the stricture was relieved. The gut was left in the sac, together with some inflamed omentum. The patient died the next day with general peritonitis, and it was ascertained that the ileum, one foot from the ileo-cæcal valve, had been ulcerated through by Gimbernat's ligament, but that owing to adhesions, fæces had not entered the peritoneal cavity, although the sub-peritoneal tissues were widely infiltrated with them.

Case 16.—*Gangrene of intestine ; death on second day.*—The patient was a woman, æt. 68, who had had a strangulated femoral hernia for six days. The sac and intestine were both gangrenous with fæcal extravasation. The ring was enlarged and the gut freely incised. The woman died on the second day and no examination was allowed.

Case 17.—*Gangrene of intestine ; death on second day.*—A man, æt. 68, had had an inguinal hernia of the right side strangulated for five days—was operated upon by Mr. Willett. The intestine was gangrenous and had given way ; it was stitched to the edge of the wound and opened.

The stricture was not divided, but stretched by introducing the finger into the intestine. There was some fæcal discharge at once, and the bowels were afterwards open naturally. He died on the second day, it was thought, of peritonitis, but none was discovered at the examination.

Case 18.—*Gangrene of intestine; death on second day.*—In 1888, Mr. Willett operated upon a woman, æt. 40, who had two femoral hernias, the right of which had been strangulated for three days. The sac contained omentum and intestine, which, being gangrenous, was laid open and stitched to the edges of the wound. She died the next day without having an action of the bowels, and with vomiting and collapse. It was ascertained that there was general peritonitis with fæcal extravasation into the peritoneum of the right iliac fossa. This had been permitted by an ulcerated aperture in the constricted part of the intestine.

Case 19.—*Gangrene and ulceration of intestine; gangrene of edges of wound; no relief; death on second day.*—In 1885, Mr. Langton operated upon a woman, æt. 55, who had had a right femoral hernia, which had been strangulated for fifty-two hours. The gut was probably gangrenous, and there was an ulcerated hole in it opposite Gimbernat's ligament. The intestine was stitched to the edges of the wound. The patient died two days later unrelieved, and the edges of the wound were gangrenous. It was ascertained that there was slight general peritonitis without any fæcal extravasation, and that the intestines above the herniated intestine were distended.

Case 20.—*Gangrene of intestine and omentum; death without relief the same day.*—A woman, æt. 33, had had a femoral hernia strangulated for three days. The sac contained eight ounces of fæculent fluid, gangrenous intestine and omentum, which was beginning to gangrene. The omentum was cut off and the wound left open, but the operation gave no relief.

Case 21.—*Ulceration of intestine; death the same day.*—A woman, æt. 59, had had a femoral hernia strangulated for two days. The operation was done by Mr. Willett, and the intestine burst as it was being returned. The incision was enlarged and the pelvis cleaned out, and an artificial anus established. The patient died soon after the operation.

Case 22.—*Gangrene of intestine; death the same day.*—A woman, æt. 60, had had a right femoral hernia strangulated for eight days. Was afterwards operated upon by Mr. Callender. The sac was full of "fœtid pus," and the gut grangrenous and ruptured. The stricture was freely incised, and the intestine, which was adherent, left to discharge. The patient died soon after the operation.

Case 23.—*Gangrene of intestine; spontaneous rupture; operation; death the same day.*—A woman, æt. 40, had some obscure trouble in a right femoral hernia of eight years' duration. Mr. Smith, under whose care she was, kept her under observation for a time, and when, at last, it was thought advisable to open the hernial sac, it was full of fæces, the gut having spontaneously ruptured. The wound was left open and the patient shortly died.

Case 24.—*Gangrenous intestine; death the same day.*—A man, æt. 42, who had congenital (infantile) inguinal herniæ on both sides, the right having been strangulated five days. The operation was done by Mr. Howard Marsh, and the intestine burst on handling, and was laid open and stitched to the wound. The patient never rallied. It was ascertained that the intestine above the hernia was much distended, and that its peritoneum was ruptured in places and almost gangrenous. There was no general peritonitis.

Case 25.—*Gangrenous intestine; death within an hour.*—A man, æt. 54, had a strangulated congenital (infantile) in-

guinal hernia of the right side. The scrotum was œdematous and discoloured, and the patient was collapsed. Mr. Morrant Baker found a foot of gangrenous intestine in the sac, and it was laid open. The patient never rallied, and died on his way back to bed.

Case 26.—*Gangrene of intestine; death same day.*—In 1886 Mr. Langton operated upon a man, æt. 62, who had an inguinal hernia of the right side strangulated for three days, and the skin over it was emphysematous. The sac contained gangrenous intestine (ileum) which was laid open and stitched to the edges of the wound. The patient shortly afterwards died, and it was ascertained that there was intra-peritoneal fæcal extravasation and peritonitis.

Case 27.—*Gangrene of intestine; death thirteen hours after the operation, apparently unrelieved; peritonitis.*—A man, æt. 57, who had an enormous left scrotal hernia which contained large and small intestine; the latter was gangrenous and was laid open and stitched to the edges of the wound. The patient died unrelieved thirteen hours after, and it was ascertained that the beginning of the ileum, twelve feet from the ileo-cæcal valve, had been stitched to the wound, and also that there was general peritonitis. The strangulation was caused by a fibrous adhesion, which crossed the fundus of the hernial sac.

Case 28.—*Ulceration of intestine; death same day; general peritonitis.*—A man, æt. 48, had had a right femoral hernia strangulated for three days, was operated upon by Mr. Butlin. The sac was full of fæculent fluid, and the intestine, which was ulcerated, opposite Gimbernat's ligament and towards the convexity of the knuckle. The patient never rallied, and it was found that the stricture had been relieved, but that there was general peritonitis. No mention is made of fæcal extravasation.

Case 29.—*Gangrene of intestine; death the same day.*—A female, æt. 62, had a femoral hernia, which was supposed

to have been strangulated eight days. At the operation it was found that the knuckle of intestine had sloughed off. The wound was left open but the patient soon died. There was no examination and the notes are very meagre.

Case 30.—*Gangrenous intestine; death in seven hours.*—A woman, æt. 54, who had had a femoral hernia strangulated for eight days. The intestine was gangrenous and was laid open. The patient died in seven hours. There was no post-mortem examination.

Case 31.—*Gangrene of intestine; suppuration in great omentum; death; fæcal extravasation and general peritonitis.*—Mr. Thomas Smith operated upon a woman, æt. 72, who had had a right femoral hernia strangulated for four days. The intestine was left in the sac, and the patient having died it was ascertained the ileum was gangrenous and also perforated at the femoral ring by Gimbernat's ligament. There was general peritonitis and pus in the folds of the great omentum. It is noted that in this case the pain ceased at what was supposed to have been the onset of the gangrene.

Case 32.[1]—*Strangulated inguinal hernia in an infant; gangrene of intestine; death in third week.*—In July Mr. Langton operated upon an infant five weeks old, who had a strangulated inguinal hernia of the right side. The hernia, which was congenital, had been strangulated some days, and contained gangrenous intestine. The constriction was dilated with forceps and the bowel incised. There was afterwards a free flow of fæces from the wound, but chronic peritonitis supervened, and the child died at the end of three weeks. It was ascertained that the wound contained a loop of ileum, which was distant three inches from the ileo-cæcal valve. The peritoneal cavity contained no fluid, but there were general adhesions, and the intestines above the wound were distended.

[1] This and the remaining cases belong to the postscript.

Case 33.—*Strangulated femoral hernia ; partial enterocele ; ulceration of intestine ; death after three days—unrelieved.*—A woman, æt. 65, came under the care of Mr. Willett with a femoral hernia of the right side which had been strangulated two days and a half. The gut was ulcerated through opposite Gimbernat's ligament, so the constriction was divided and the bowel left in place, the edges of the rent being stitched to the edges of the wound. There was no fæcal flow from the wound and the sickness continued, the abdomen being tender and tympanitic. Flatus, however, was passed per anum, and the bowels were slightly open. She died after living three days, and it was ascertained that only a part of the lumen of the bowel (twelve inches from the ileo-cæcal valve), was involved in the hernia, but that the intestines above it were distended and inflamed, whilst below they were flaccid and empty. There was no fluid or fæcal matter in the peritoneum.

Case 34.—*Right femoral hernia ; ulceration of intestine ; rupture of intestine ; fæcal extravasation ; incision ; death without relief.*—A woman, æt. 54, had had a right femoral hernia strangulated for some days. The intestine ruptured after it had been reduced owing to ulceration at the line of constriction, opposite Gimbernat's ligament. It was drawn down, opened freely, and stitched to the edges of the wound by Mr. Long (Sir William Savory's House Surgeon). The stricture was divided at the early stages of the operation. The patient had no relief by the incision, and is said to have died of shock thirty hours after the operation, and it was ascertained that there was general purulent peritonitis with some fæcal extravasation. A loop of ileum three feet from the ileo-cæcal valve was adherent in the wound.

Case 35.—*Strangulated femoral hernia ; partial enterocele ; gangrene of intestine ; death in a few hours—unrelieved.*—A woman, æt. 86, who had had a femoral hernia

strangulated for four days, was operated upon by Mr. Willett. The bowel was gangrenous, and was laid open and stitched to the edges of the wound. The patient survived some hours but had no relief. It was ascertained that only a part of the lumen of the intestine was involved in the hernia, but that there was enormous distension above the opening, but no extravasation of fæces. The peritoneum and intestines were considerably injected, and the sac seemed to be gangrenous.

Case 36.—*Strangulated femoral hernia; intestine and sac gangrenous; death during operation.*—A woman, æt. 53, had had a right femoral hernia strangulated for three days. She was exhausted and collapsed on admission, and died as the operation was being completed by Mr. Holden (Mr. Willett's House-Surgeon). At the operation the sac was gangrenous, and the intestine ashen-grey and perforated in two places. This case has merely been referred to in the body of the paper.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 102.)

ON MEASLES AS A CAUSE OF ENDOCARDITIS;

BEING AN ACCOUNT OF FOUR CASES IN WHICH ORGANIC MITRAL MURMURS DEVELOPED DURING THE COURSE OF THE DISEASE.

BY

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THE occurrence of endocarditis in the course of an attack of measles is either a rare complication, or one that is very generally overlooked. Cases have indeed been noted by several observers in Germany, and by Dr. Sansom in this country, but endocarditis is not even mentioned in connection with measles in any of the text-books of medicine with which I am acquainted. I hope, therefore, that having met with four cases complicated with endocarditis during a recent outbreak of measles, I am justified in placing my experience on record.

In a large preparatory school a boy fell ill with measles a few days after his return. He was immediately isolated, but between twelve and twenty-two days afterwards no

fewer than fifty-eight out of eighty-five boys contracted the disease. The outbreak was on the whole of a mild type; few of the cases were complicated, and all finally recovered.

The boys were all in one house, which was well situated, recently built, and furnished with the most perfect sanitary appliances; they all belonged to the upper middle class, and were well cared for in every way. Four of those who contracted measles developed cardiac symptoms during the course of the attack.

Case 1.—A delicate looking boy, æt. 10, of a very rheumatic family, one brother having died of valvular disease of the heart. The boy had been examined by the family medical attendant previous to his leaving home a fortnight before, and pronounced free from disease. He is stated to have had chorea, but never to have suffered from articular rheumatism.

He was first examined by me on the second day of the prodromal stage, when he was suffering from catarrh and fever, the temperature being 103·5°, the pulse 116.

On listening to the heart I found a well-marked mitral systolic murmur audible over the apex, and conducted towards the axilla.

On the following day the pulse had become irregular, and in addition to the systolic apex murmur, a præsystolic murmur was audible over a limited area midway between the apex-beat and the left edge of the sternum; the second sound was accentuated in the pulmonary region, and reduplicated.

On the fourth day the temperature and pulse fell to normal in the morning, but rose again towards evening, when the rash appeared.

From this time the measles ran the usual course; the cough was rather troublesome, but there were no signs of broncho-pneumonia. The præsystolic murmur continued to be heard until the twelfth day, after which it could no longer be made out; but the systolic bruit persisted,

changing in character from day to day, yet always present during the fortnight the boy remained under my observation. Six weeks later I was informed by the family medical attendant that this murmur had also disappeared, but the pulse was still irregular, and intermitting, 1 in 40, and the boy remained anæmic.

Case 2.—A boy, æt. 9, stated to have had croup, but I could obtain no history of rheumatism. The measles was of only moderate severity until the fourth day of the eruption, when a severe attack of catarrhal croup came on, and fine râles with bronchial breathing and some dulness on percussion were observed over limited areas at the bases of both lungs.

These signs gradually subsided, and all appeared to be going well. The heart had been examined daily, but the sounds remained normal until the fifteenth day of the disease, when the pulse became slightly irregular, and a systolic murmur was heard over the apex-beat, and conducted towards the axilla. This caused no subjective symptoms; convalescence has slowly progressed, and the boy considers himself quite well. The murmur, however, is still distinctly audible.

Case 3.—A small delicate boy, æt. 9, subject to bronchitis, but he has never had articular rheumatism. Several members of his family are said to have suffered from heart disease; his father dropped down dead at the age of forty-two; his paternal uncle and grandfather and several relations on his mother's side are reported to have had heart complaints, but it is not known whether they were of rheumatic origin.

On the second day of the rash fine crepitations with bronchial breathing and dulness on percussion were observed in patches scattered over both lungs, with pleuritic friction over a considerable area on the left side, followed later by signs of effusion. The respiration was quickened, and the temperature went up. The heart's sounds were

clear. On the fourth day the pulse became irregular and very intermittent, and on auscultation a systolic murmur was audible at the apex, with a præsystolic murmur also a little more to the right, near the left edge of the sternum.

During the following three weeks the pulmonary symptoms gradually disappeared. The heart signs varied from day to day, the pulse being at times very irregular and infrequent, falling to 48 beats per minute, and at other times ranging from 70 to 80, but never regular.

Six weeks after the commencement of the attack both the murmurs were still present, and some hypertrophy had taken place, indicated by the fact that the apex-beat had advanced to the nipple line. The boy was not conscious of any cardiac discomfort.

Case 4.—A boy, æt. 11, commenced with the usual catarrh, and, in addition, rather severe tonsillitis. The tonsils were evidently hypertrophied, and had suffered from previous inflammatory attacks. The boy had never had articular rheumatism, nor was there any history of family predisposition. The rash, which was abundant, and the cough, which was extremely troublesome, disappeared in the usual course, and convalescence seemed to be well established, when on the fourteenth day the pulse became irregular, and a soft blowing systolic murmur was heard at the apex for the first time. The heart had been frequently examined previously, and its sounds had been perfectly normal. This complication was not caused by a chill, as the boy had never left his room. Signs of subacute broncho-pneumonia also developed, but the rise of temperature was very slight. The cough remained troublesome for some weeks. The murmur altered considerably in character, and became less loud, but at the end of seven weeks, when the boy passed from under my observation, the mitral regurgitant bruit was still distinctly audible.

Remarks.—The development of endocarditis during the course of the attack of measles in these cases was no doubt

directly due to the effect of the measles poison. But it becomes a question how far an hereditary rheumatic taint may have prevailed in inducing the heart complication. In Case 1 there was a strong rheumatic history, and although the boy had never had articular rheumatism, he had suffered from chorea. The patient in Case 3, in whose family so many instances of heart disease occurred, was probably of rheumatic ancestry; and in Case 4 the boy had had repeated attacks of follicular tonsillitis. None of the cases presented any symptoms of articular rheumatism whilst under my personal observation.

It will be noted that the endocarditis developed in one instance (Case 1) during the prodromal stage; in another (Case 3) during the height of the measles, and in the other two (Cases 2 and 4) during convalescence, viz. on the fifteenth and fourteenth days respectively.

In none of the cases were there any subjective symptoms; there was no palpitation or pain, or dyspnœa; and the diagnosis was founded entirely upon the physical signs, viz. the irregular pulse, the apex murmurs, and the accentuated and reduplicated pulmonary second sound. I am aware that these signs have been stated to be sometimes present where myocarditis alone existed, but in these cases I cannot interpret the physical signs otherwise than as evidence of organic valvular mischief. The persistence of the murmurs, especially of the præsystolic in Case 3, the accentuated and reduplicated second sounds over the pulmonary area, and in one case (Case 3) the supervention of hypertrophy, are, I think, conclusive on this point. There were no physical signs of pericarditis in any of the cases.

I may state that in all these cases of measles the condition of the heart and lungs was examined daily, and my experience leads me to believe that if this rule were more generally observed, we might find, as Dr. Sansom affirms in his Lettsomian Lectures, "that the influence of measles in predisposing to endocarditis, has been much underrated."

(For report of the discussion on this paper, see ' Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 112.)

ON THE VALUE OF TUBERCULIN

IN THE

TREATMENT OF SURGICAL TUBERCULAR DISEASES.

BY

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In considering the subject under discussion to-night it will, perhaps in some ways be most convenient, if I commence by referring to the modes in which the remedy has been employed in my cases, and to those which I believe to be most likely to yield good results. For experience has convinced me that the ultimate result depends to a great extent on the mode in which the fluid is used, and that the method of administration must vary to some extent with the aim which one has in view. I will only refer in detail to two methods. The first, which may be spoken of as the "intermittent" method, is that which was recommended by Dr. Koch, and which has been followed by most of those who have used this treatment: the great majority of the cases in my list have been treated in this way. The guiding point in this plan is the occurrence of febrile reactions. The recommendation is to begin with a certain dose, and if febrile reaction

occurs, repeat the dose when the fever has subsided; if after the second dose there is still febrile reaction repeat it again, and go on in this way till reaction is slight or absent. Then raise the dose, and continue to do so every day till reaction is again obtained, when that dose is adhered to again till there is no further elevation of temperature, and so on till one decigramme is reached. A few doses of one decigramme are then given, and if no reaction occurs the treatment is considered to be practically at an end, but a decigramme is given every week or ten days for a few weeks. There are several objections to this line of treatment. In the first place, by making the occurrence of febrile reactions the guiding point in the treatment the patient is subjected for several weeks to a recurring fever with all its disadvantages, and without any corresponding advantage. Another objection is that, as the later injections only produce very little local reaction, if they are only given every day or two the effect is spasmodic instead of being constant. A third point to which I take exception is that the treatment was said to be practically at an end when the dose of one decigramme is reached. This view was no doubt based on the idea that the cessation of reaction implied complete destruction of the tubercular tissue and absence of tubercle bacilli from living tissues, and not the establishment of tolerance; but this view is, as will presently be evident, quite erroneous.

The occurrence of fever is not at all essential to the treatment; it is an unfortunate complication, at any rate in the early stage; and if it could be got rid of or modified without interference with the local effect a great advantage would be gained. The fever can be very much diminished by the administration of antipyrin in ten or fifteen grain doses two and three hours after the injection, as was first pointed out by Rosenbach, and I have found that under these circumstances the local reaction occurs and is well marked, even though the temperature remains low. Fever can also be avoided to a great extent by the second or "con-

tinuous " method of treatment which I am now using, and which I shall now describe. Careful clinical investigation shows that tolerance is much sooner established as regards febrile than as regards local reaction; in lupus, for example, it can be seen that after doses which produce no fever increased scaling occurs. This being so, it occurred to me very shortly after I commenced the treatment that it would be a good plan to try to establish tolerance as regards the febrile reaction as quickly as possible, and at the same time to try to make the local effect as continuous as we could. To do this I adhere to the original dose till no further reaction occurs with it, and then increase its frequency up to twice or thrice daily. After a few days the dose is raised, and if no reaction occurs this second amount is continued twice or thrice daily for several days, when a further increase is made, and so on (see Case No. 8). For example, in external tuberculoses I now usually commence with two milligrammes. In most cases this produces a febrile reaction which seldom passes off completely in less than thirty-six or forty-eight hours. After forty-eight hours, if the fever has subsided, I repeat the dose; again fever occurs, and again it may be necessary to wait forty-eight hours. At the end of that time the same dose is injected, and now in many cases the fever will be less, and will subside within twenty-four hours; if that is the case the dose is repeated at the end of that time. Usually after two or three daily doses the rise of temperature and the general symptoms are insignificant or absent, and in that case I give it twice, and later thrice daily. After a few days the dose is increased, and in this way the amount may be carried up to several decigrammes thrice daily without the patient experiencing any bad effects. Further, the treatment should be continued for a long time. Proceeding in this way, the febrile or disadvantageous part of the treatment is got over in a week or ten days, while for a long period the local action is kept up, as evidenced in lupus for example, by continued redness and scaling and steady

improvement. And not only have cases treated in this way from the first improved markedly, but cases which had come to a standstill under the ordinary method are now improving under this plan. At first sight this method may appear to be an heroic one, but it is not really so. The patients treated in this way have suffered much less than those treated by the earlier plan. The initial dose and the rapidity of increase must of course vary in each case; the former may be small or large, the latter slow or rapid, according to circumstances and the results aimed at. I lay more stress on the frequency than on the amount, and from what I have seen I am inclined to think that treatment in this way will do more good and less harm than by Koch's original method.

There are various other plans which might be tried, such as large doses at long intervals, so as to avoid the establishment of tolerance, very small doses every few days, and so on, but they do not seem to promise any advantage; indeed, the last plan seems to me distinctly bad.

With regard to the phenomena which occur after the early injections, so much has already been written that it is needless for me to go over the ground again, and I shall therefore pass on to the points raised in this paper. These points are three in number, viz. the dangers of the treatment, its capabilities, and its place in surgery.

Dangers of Tuberculin.

Taking first the dangers of the treatment, we may consider them under two headings, viz., 1, dangers due to the action of the remedy on the body generally—poisonous effects; and 2, dangers in connection with the action of the remedy on the affected parts.

As regards the dangers due to the action of the remedy on the body generally, I have in two instances, where the doses employed were large, observed poisonous effects following immediately after the injection. In the one

case (No. 2) I had gradually raised the dose up to 4½ decigrammes, and the occurrence to which I refer took place after the second injection of this dose. About ten minutes after I had left the house Dr. Thin was sent for, the patient stating that a few minutes after the injection she had felt very ill, and a rash had come out. Dr. Thin found the patient considerably collapsed, with a weak intermitting pulse, and covered with urticaria. These symptoms soon passed off, and when I saw her about six hours after the injection she had quite recovered. In the other case I happened to come into the ward at Paddington Green Children's Hospital at the time that the house surgeon was giving the injections, and I found him at the bedside of a little child (No. 22) to whom he had just given a dose of 3½ decigrammes. He told me that about half a minute after the injection the child began to cry loudly, and then rapidly became cyanosed. When I saw her the skin all over the body was deeply cyanosed, and the pulse was very feeble, but the child was crying strongly. Warm bottles were applied, and within five minutes the cyanosis had disappeared, and the pulse had much improved. In these two cases it seems to me that the fluid must have been injected directly into the circulation, for in both as large doses had been administered before without any such result; and besides, the immediate onset of the symptoms implies immediate entrance of the fluid into the circulation. These cases teach us that great care must be employed in using large doses if it seems necessary to do so, and I believe the risk can be avoided by moving about the point of the needle in the subcutaneous tissues so as to form a pocket, and by injecting the fluid very slowly.

A much more common period at which danger may arise is during the early febrile reactions, and the most alarming general symptom is the cardiac depression which is frequently present to a slight extent, but sometimes, especially where the first dose is large, to such a degree as to cause anxiety: this depression is often most marked

during the fall of the temperature. In only one of our cases was this depression so great as to cause alarm, viz. in the leper (No. 56). About six hours after the third injection the patient passed for a short time into a more or less collapsed condition, with a very slow and weak pulse. I have not been able to find any recorded case in which death occurred as the result of this cardiac depression, although several patients have evidently been in a very critical condition. Indeed, I can only find a record of two cases which died during the early stage of the treatment. One of these was the Innsbruck case which has been mentioned in the journals. The case was that of a female æt. 17, with extensive lupus about the face, nose, mouth, &c., but, so far as was discovered on clinical examination, with no internal tuberculosis. On December 3rd, at 9.30 a.m. two milligrammes were injected for the first time. This was followed by marked local and general reaction, the temperature fifteen hours after the injection reaching 106° F. Next morning the patient was very drowsy, the pulse intermittent, weak, and very rapid (from 150 upwards), respirations 40 ; the somnolence continued, and fæces were passed in bed. During the afternoon there were twitchings and convulsions, and the patient became quite unconscious and died thirty-six hours after the injection. On post-mortem examination there was marked œdema of both lungs, and numerous patches of pneumonia scattered through them ; extreme œdema of the brain and spinal cord ; capillary hæmorrhages under the pleura and pericardium, and into the thymus and spinal cord ; inflammatory changes in connection with the tubercular lesions on the skin, in the glands, and in various parts of the intestine. The second case is reported by Schmid ('D. M. W.,' 1891, No. 1), and was that of a boy æt. 5, whose knee-joint had been excised six months previously, and whose general and local condition was bad. He did not react after 2 and 4 milligrammes ; on the sixth day 6 milligrammes were injected ; two hours later there was a slight rigor, and the tempe-

rature had risen to 102° F. Severe cerebral symptoms commenced about the same time; there was twitching of both sides of the face, Cheyne-Stokes respiration, and death fifteen hours after the injection. On post-mortem examination there was great hyperæmia of the brain, with about twelve hæmorrhages the size of sixpence scattered over it and penetrating for half an inch or more into its substance; there was also thrombosis of one of the sinuses, which, however, was of older date. Schmid leaves it an open question whether the hæmorrhages were due to the drug or to the previously existing thrombosis.

When we take into account the very large number of patients who have now been subjected to this treatment, and the fact that the cases where death has occurred during the febrile period are so extremely few, we must, I think, conclude that the risk of death as the immediate result of the injections is not one which need deter us from employing the treatment. The risk is not nearly so great as that of many surgical operations for tuberculous disease.

In connection with the immediate risks of the treatment regard must be had to the possible cumulative action of the remedy. We not unfrequently observe that if the second dose is followed quickly on the first the temperature is higher, and sometimes also the constitutional symptoms are more severe. This is especially the case when the second injection is given before the temperature has completely fallen after the first. This happened in the first case which I injected (No. 1), where the first reaction was severe and the temperature was slow in falling. On the fifth day after the first injection, while the temperature was still 100° F., I repeated the dose, and the result was a much more severe reaction than at first, and indeed for a day her condition was such as to cause us considerable anxiety. (I may say that this case and the leper were the only two which gave us any anxiety during the reactions.) These cumulative effects are only, as a rule, seen during the very early injections.

At a later period of the treatment tolerance is being established, and this cumulative effect is not apparent. In only one instance have I observed it to any appreciable extent at a late period in the treatment, and in that case tolerance had not yet been established (No. 52). In the continuous method of administration which I advocate, and where we would expect this effect to be most marked, we usually see nothing of it, though in some cases there may be a slight rise of temperature after a few doses have been given; this, however, is generally where we begin to repeat the dose frequently before reaction at that dose has quite ceased.

The other general effects, such as anæmia, loss of weight, &c., need not be considered here, and I may pass on to the second group of dangers, viz. those due to the action of the remedy on the tubercular tissues.

In the first place great stress is now being laid by some on a supposed risk of dissemination of the disease, and that in two situations, viz. either generally throughout the body, or locally in the immediate vicinity of the original deposit. In reference to the occurrence of acute general tuberculosis, we know that this only takes place when a quantity of tubercular material is poured into the circulation more or less suddenly, generally from involvement of the wall of a vessel in the tubercular growth, subsequent destruction of the wall, and communication of the tubercular material with the still patent lumen of the vessel. A few bacilli passing into the blood will not suffice to produce general tuberculosis, or indeed of themselves to set up a secondary deposit; and in my recent lectures at the College of Surgeons I referred to evidence which shows that bacilli are frequently present in the blood in cases of tuberculous disease without giving rise to any local lesions. Indeed, if the bacilli floating in the blood are only few in number, they will, in all probability, be quickly destroyed, more quickly than in the tissues. Now there is no evidence whatever that after the injection of tuberculin the bacilli get into the blood in un-

usual numbers in any other way than by the ordinary modes of communication of a tubercular deposit with the circulation. Nauwerck alone has put forward the view that after the injection of tuberculin the bacilli grow more rapidly, and that the larger numbers of bacilli found in the sputum during the early period of the treatment are to be explained by increased growth, and not, as appears at first sight more probable, by more rapid passage of the organisms into the sputum. This view is not supported by evidence so far as surgical tubercular diseases are concerned, for in lupus, tubercular glands, and the synovial membrane of joints I have found it just as difficult to find bacilli after Koch's treatment as is usually the case: there is no sign in these instances of more rapid multiplication of the organisms. Liebmann's statement that he has found numerous tubercle bacilli in the blood of phthisical patients undergoing Koch's treatment is absurd on the face of it, for even in the most virulent infective disease the micro-organisms in the blood are seldom so numerous as they appear to have been according to his statement and in the most acute general tuberculosis it is practically impossible to demonstrate tubercle bacilli in the blood by means of the microscope. Several trustworthy observers have since examined the blood of patients under this treatment, and have invariably failed to find bacilli; while Ehrlich, Guttmann, Kitasato, and Kossel examined Liebmann's original specimens, and found that they had been contaminated with phthisical sputum, and that it was in the sputum and not in the blood that the bacilli were present.

Where a tubercular deposit is present which involves the wall of a blood-vessel it is not at all improbable that the effect of the tuberculin may be to establish a communication with the interior of the vessel more quickly than would otherwise have occurred; but where this condition is present the patient is already in imminent risk of acute tuberculosis quite apart from Koch's remedy, and it is most difficult to estimate whether there is any or

how much increased risk of this occurrence after the use of this material. Indeed, it is equally probable that under the above circumstances the effect of the tuberculin may be to save the patient from the impending general disease, in that it may arrest further growth of the tubercle, or in that the inflammation around the deposit set up by the remedy may lead to thrombosis of the vessel, and thus shut off the deposit from the general circulation. We can only arrive at any sort of conclusion on this matter by the careful comparison of extensive statistics of cases treated by tuberculin, and of similar cases treated in other ways, and this has not yet been done. So far as I can see from what has been published on this subject, the increased risk of general tuberculosis from the use of this remedy must be very small. To put down every, or indeed any case of acute tuberculosis which occurs during this treatment to the effect of the remedy is at present utterly illogical, and is only to fall into the *post hoc ergo propter hoc* fallacy, that great bugbear of scientific medicine. I may illustrate how this fallacy would tell in the opposite direction from my own experience. When I commenced the treatment at the end of November, I made up my mind that in order to ascertain the capabilities of the remedy, I would not attempt to select cases at first, but would admit and treat any patient who applied till my wards were full. This resolution I carried out with two exceptions ; one of these cases was a man who was already in the ward before I went to Berlin, and who had consented, and indeed was anxious to undergo the treatment immediately on my return. It was a case of spinal disease and early tubercular disease of the kidneys. When I came from Berlin I found that the patient had developed some obscure lung trouble with high temperature, and I therefore sent him up to the medical wards, intending to carry out the treatment at a later period. The other was a patient with extensive disease of the sacrum, and of one hip-joint with septic sinuses, who was admitted at the end of December.

I had already by that time become chary of employing the treatment where there was a septic complication, and I therefore did not use it in this case. Both of these patients died of acute general tuberculosis during January, the lungs in the first case especially being completely infiltrated with miliary tubercles. Now of fifty-five cases of tubercular disease in which I have employed this treatment none have developed acute general tuberculosis, while the only two cases in which I declined to use it died of that disease. Allowing the *post hoc ergo propter hoc* fallacy to come into play, we should conclude that the turberculin prevented the occurrence of general tuberculosis in the cases treated—a conclusion as unwarranted as the opposite one, that where acute tuberculosis occurs in the course of the treatment it is therefore due to it.

More important because more probable is the question of the risk of dissemination of the disease in the immediate vicinity of the original tubercular deposit. This is, I suspect, much more likely to occur in internal than in external tuberculosis, and the possibilities are undoubtedly greatest in disease of the lungs. In the first place, if the local reaction is considerable, and a large amount of fluid is poured out, carrying with it the bacilli and débris from the surface of a cavity, there is the risk that this increased quantity of material may not be properly expectorated, and that portions may be inhaled into other parts of the same lung or into the other lung, and there set up further disease. This is a danger which must be guarded against by taking care that the reaction shall not be excessive.

A second way in which dissemination may occur in the case of the lungs is where the disease is situated close to or involving the pleura, and where adhesion of the two layers of the pleura has not taken place. Under these circumstances it may quite well happen that after the injection, ulceration or destruction of the thin tissue separating the tubercular deposit from the serous cavity may take place, and the tubercular virus be poured into the cavity, caus-

ing infection and growth of the endothelial lining of the pleura, which being at first non-vascular would go on growing quite unaffected by the remedy, and so produce the masses of tubercles which have been described. A similar occurrence may also take place in connection with other cavities, such as the peritoneum and the joints. In the case of joints it is quite possible, and has indeed apparently happened, that where an osseous dsposit has not yet burst into a joint, but is on the verge of doing so, the occurrence may be precipitated by the use of tuberculin.

A third way in which it is conceivable that local dissemination might occur is, that the tubercular area being distended during the early reactions by a large quantity of effused fluid, bacilli may be floated away by this fluid and deposited in neighbouring parts, and there set up new growth of tubercular tissue. That a dissemination of the disease can occur in this way in the immediate vicinity of a tubercular patch under the ordinary method of treatment seems not impossible from what has been found in some post-mortem examinations, and from what has been observed clinically, especially in the larynx. At the same time I cannot think that this will be other than an exceptional occurrence, seeing that the tubercle bacilli are usually present in the fixed cells, and would not, therefore, be easily carried away; while, as a matter of fact, bacilli have not been found beyond the tubercular areas. The idea arose at a time when Koch's view that the treatment caused caseation, and thus might free the bacilli, was accepted; but this view is incorrect. If dissemination can take place in this way, clearly the best way of meeting it is the continuous method of administration, by which the new tubercular tissue cannot increase to any extent before it is attacked by a fresh dose. Under the intermittent method, more especially when small doses are given at considerable intervals, the new tubercular tissue could develop practically without interruption.

So far as my own experience goes, I have had no

example of general dissemination of tubercle under the treatment, and case No. 46 is the only one which lends any support to the view of local spread.

Apart from the poisonous action of the substance, and the possibility of dissemination of the tubercular virus, there are other dangers to which I must allude. The first is from the inflammation which is set up around the tubercles. As is shown both by clinical and microscopical observation, the effect of the early injections is to cause distinct and, according to circumstances, even violent inflammation around the tubercular area. This inflammation is not, however, of a spreading character, and is quite transient, passing off within a few hours, and consequently in external tuberculosis it is not associated with any special danger. It is, however, evident that this inflammation, if severe, may be attended with serious consequences, especially in extensive internal disease, as in the lungs and kidneys. The remedy for this is to begin with small doses.

What in my opinion constitutes the greatest danger in connection with the treatment, and one of far more importance than any to which I have as yet alluded, is the conjunction of sepsis[1] with the tubercular process; this danger is, of course, common to medicine and surgery. Where this combination exists, whatever may be the effect of the tuberculin on the tubercular tissue itself, it certainly seems to aid the extension of the septic process, and that in various ways. In the first place, during the early injections, a quantity of serous fluid is poured out into the sinus or cavity in which the pyogenic organisms are present, and thus provides them with pabulum for their growth; this is readily seen in the increased suppuration which follows the use of the injections in these cases. In the second place, the transient inflammations of the wall of the cavity weaken the tissue, and thus favour the penetration of the pyogenic organisms into it,

[1] Under the term sepsis I refer only to those forms of disease due to the pyogenic organisms and their allies.

and the consequent extension of the suppurative process. And, thirdly, I cannot but think that not only does tuberculin produce conditions favorable to the local extension of the septic process, but that the body as a whole becomes more prone to septic infection.[1] Several instances have now been published in which erysipelas has followed injections in cases of lupus; and as regards sepsis in other parts of the body, I have seen several things which have made me chary of employing this treatment in septic cases. Take, for example, No. 32. This patient had two septic sinuses, one in the lumbar region connected with the spine, and one in connection with disease of the left elbow-joint; and she also had tubercular disease of the occipital bone, with a semi-fluctuating swelling over it, which had not yet been opened. After a few injections this swelling over the occipital bone became so much smaller that it was only found with difficulty, and the tenderness became less. On the other hand, the sinus in the back did not improve, the elbow became much more painful and swollen, and her general condition became worse. In this case, in the part where no sepsis was present, there was improvement as regards the tubercular process, and the bad result as regards the elbow was evidently due to the septic condition rather than to any unfavorable action of the remedy on the tubercular disease. Take again No. 42, a child with hectic temperature and with septic sinuses in the groin in connection with tubercular glands. After a few injections the temperature became continuously high, there was burrowing of pus in the left iliac fossa and groin, and marked increase in the amount of discharge; a communication also formed with the left hip-joint, leading to an acute septic arthritis of that joint.

I believe that the danger where sepsis is present, due to extension of the septic rather than of the tubercular

[1] With regard to this point I have made some experiments on animals by injecting tuberculin and inoculating pyogenic organisms, but have obtained no evidence confirmatory of this view (July).

process, is a very real one, and much greater than that of dissemination of the tubercular disease, which is, after all, only problematical. In most of the published cases in which continuous fever has supervened on the injections the patients have had suppurating sinuses or cavities, and here the extension of the septic process is, I believe, the true explanation of the phenomena. So real, in my opinion, is the danger in these cases that I should be inclined to exclude from the treatment most of the cases, whether of external or internal tuberculosis, in which this complication is present, or at any rate not to treat them till a very free exit had been provided for the escape of the pus, and an attempt had been made to get rid of the septic element.

To sum up what I have to say with regard to the dangers of the treatment, I believe that the great danger is the combination of sepsis with the tuberculosis; the other dangers can, I think, be for the most part avoided by care, and are not such as to deter us from using the treatment in cases where otherwise it is likely to be of value.[1] Whatever the dangers and disadvantages be, they must be weighed against the advantages in any given case, just as in deciding on operative interference in tuberculous disease we have to weigh the dangers of dissemination of the disease, of shock, of possible sepsis, the disadvantages as regards the growth of the limb in joint disease, &c., against the probable advantages. We must therefore now inquire what this substance can do in the way of cure or arrest of the disease; and it is on the answer to this question rather than on the possible dangers that the future of the treatment depends.

[1] Another danger of the treatment which has attracted my attention lately is that tuberculin seems to render the body more susceptible to tubercular infection, instead of producing immunity, and therefore, if a cure is not obtained before leaving off the treatment, very rapid recurrence will probably take place, and the condition of the patient become worse than before (July).

The Capabilities of the Treatment.

The question as to the capabilities of the treatment must be decided on evidence obtained from clinical observation and from pathological investigation, and it will perhaps be most convenient if we begin with the latter evidence.

When we examine microscopical sections of tubercular tissues which have been subjected to the action of tuberculin, we see that very considerable changes have occurred in connection with the cellular elements. The first effect of the remedy is to cause a transient but acute inflammation in the tissues immediately surrounding the tubercles; and thus in sections made a few hours, or, better, a day or two after the first injection we find a great increase in the number of the leucocytes, which are in parts commencing to penetrate into the tubercles, and we also see evidences that the tissues have been more or less distended with exuded fluid. These acute inflammatory changes recur in a lessening degree after the earlier injections, and at a later period the appearances observed indicate an absorptive and healing process.

If we examine tubercular tissue from patients who have been under treatment for six or eight weeks we can note the following points. In the first place, looking at the essential cellular elements of tubercle, the epithelioid and giant cells, we see that they have undergone grave changes as regards their nutrition, and many of them are evidently dead, and simply appear as clumps of degenerating protoplasm in which the nuclei are not at all visible or only very faintly stained. The epithelioid cells almost always suffer before the giant-cells, and it is not uncommon to find in a tubercle that the epithelioid cells are more or less shapeless masses, while the giant-cells are fairly well preserved. At this time the tubercles are becoming infiltrated with small cells, and the remains of the epithelioid cells are disappearing before them, so that

in several of the photographs exhibited the place of the tubercle is taken by a mass of round-cells, scattered throughout which we find the remains of the epithelioid and giant cells. In addition to the small cells which apparently spread in in the first instance we not unfrequently find larger cells following them, and thus obtain appearances recalling very strongly Ballance and Sherrington's description of the changes which occur in the organisation of lymph. The next step is the development of fibrous tissue from these cells (not, so far as I can see, from the epithelioid cells, as some German writers have asserted), and the ultimate result is the disappearance of the tubercle and its replacement by fibrous tissue.

In some cases the giant-cell persists for a long time, and may be found lying in this newly formed fibrous tissue. The changes thus consist in transient inflammation around the tubercles at an early stage, accompanied by serious damage to the tubercular cellular elements, ending in many cases in their death. This is followed by absorption of these degenerated cells, and their replacement by young fibrous tissue. These changes are in all probability similar to those which occur in the natural cure of tubercle.

I have placed a number of specimens showing the various changes under the microscopes, and although the matter does not lend itself very well to photography, as the staining effects are of great assistance, nevertheless I think that I can illustrate my meaning by a few photographs which have been made for me by Mr. Pringle, of specimens illustrating the later changes.[1] (See Plates III, IV, and V.)

It will have been observed that the specimens which I have shown are all taken from isolated tubercles embedded in vascular connective tissue. It is in these that the changes which I have been describing are most manifest,

[1] Here several photographs were shown, from some of which the plates have been prepared.

and this would naturally be the case, seeing that the blood-stream is in close relation to them. Where there are closely aggregated masses of tubercles the changes are not as a rule nearly so marked ; indeed, some, especially of the more centrally placed ones, may appear quite unaffected after six or eight weeks' treatment. It thus seems that outlying and isolated tubercles may be completely destroyed by the treatment, but that where the tubercles are collected in masses portions are very likely to remain and act as foci of new growth when the treatment is left off, or when local tolerance has been so completely established that the remedy has lost its action. It is also not improbable that in some cases the increased infiltration of these masses of tubercle with round-cells may not lead to retrogressive changes, but to the formation of a chronic abscess. The recognition of these histological facts is very important in regard to the questions of relapse and of combination with surgical measures.

As regards the bacilli, the material apparently does not act on them directly, but only indirectly by getting rid of their surroundings and bringing them into contact with healthy, living, vigorous tissues, in which they will succumb more or less quickly. Where there are masses of tubercular tissue the bacilli will also remain and grow on the cessation of the treatment. I have tested all the tissues which I have removed from patients who have been subjected to this treatment by inoculating two guinea-pigs with the tissues from each case. Of eleven experiments of this kind, ten have yielded positive results, the animals becoming tubercular after the usual period of time. The length of time for which these patients had been under treatment varied from thirty-four to sixty-eight days. The case where a negative result was obtained had been under treatment for thirty-four days, and numerous tubercles in various stages of decay were seen under the microscope.

As regards experiments on animals, I have, in conjunction with Mr. G. L. Cheatle, been treating the animals

just spoken of with tuberculin, but our experiments are not yet in a fit state to publish. I may, however, say that in all cases the ulcers at the site of inoculation have healed under treatment, and the enlarged glands in the vicinity have diminished markedly in size, this diminution only lasting, however, so long as the treatment is continued. Further, in the animals which have died, the disease in the internal organs has been slight as compared with the usual amount found in animals which have lived for the same length of time after inoculation. In some, indeed, as in the case exhibited, there was no internal tuberculosis, although the treatment was begun at a period (after fifty-three days) when internal tuberculosis is usually evident, and although the companion animal killed when the injections in the other were commenced showed signs of commencing tuberculosis in the lungs and spleen. In this animal, on the other hand, tubercles are present in the lungs, and have no doubt enlarged somewhat during treatment, but there is no sign of fresh tubercle formation. In other two cases, again, there must have been fresh development of tubercles going on slowly during treatment, but in these the dose was small and was not increased. I cannot go further into this matter, but will merely show the organs of the rabbit, which was killed this evening.

This rabbit was inoculated from the case of iris tuberculosis (No. 44) on January 7th, the inoculation being made into the anterior chambers of both eyes. Twenty-six days later, there being well-marked signs of tubercular iritis in both eyes, the right being much more advanced than the left, the treatment was commenced. The first dose was 1 centigramme, and the amount was gradually increased to 3 decigrammes, at which it has remained for some weeks: sixty doses have now been given. In spite of the treatment the right eye has got steadily worse, and is now a caseous mass; the left eye remained fairly stationary for some weeks, but of late the disease seems to have progressed to some extent. We here see that the

right eye has become converted into a caseous mass. In the left eye the cornea is still clear, but there is distinct wrinkling of the iris and some nodules in it. There are several small tubercles in the lungs, but not what one would call an extensive infiltration. These tubercles are rather larger than usual, and may have been there originally, and perhaps have grown somewhat of late. No visible disease in the other organs.

To contrast with this I may quote the post-mortem appearances of one of the animals which lived longest after inoculation into both eyes, from my paper on tuberculosis published eight years ago. This animal (Exp. xiv, No. 1) lived sixty-six days (the present rabbit has lived 111 days). Eyes tubercular, cheesy glands below jaw, very extensive tuberculosis of the lungs, tubercles in liver, kidneys, spleen, large intestine, and omentum. Enlarged and caseous mesenteric and lumbar glands, &c.

On the whole the tendency of our experiments is to confirm Koch's statements as to arrest of the disease in animals by the use of this material, this arrest, however, only continuing so long as the treatment is carried on (experience of two months); and not only is there arrest in the growth of tubercles, but arrest in their dissemination.

Turning now to the clinical evidence, we find that the effect of the first injections is to cause increased swelling and pain at the seat of disease, due, as we have seen, to the inflammatory changes in the tissues immediately surrounding the tubercles. This effect is less after the subsequent injections and is soon hardly evident, and the increased swelling and pain give place as a rule to diminution of the pre-existing swelling and tenderness. In all my joint cases with unbroken skin (except No. 16, where operation was resorted to early) this improvement went on to a greater or less degree for three or four weeks, and joints which were previously rigid and tender became moveable, and in some cases quite free from pain, while they also diminished considerably in size. The meaning of this

early improvement is a point of very considerable importance, and the following is my interpretation of it. As microscopical examination shows, the thickening of the synovial membrane in cases of tubercular disease is not so much due to actual new formation of a mass of tubercular tissue as to chronic inflammation in the parts surrounding the tubercles; and the pain and tenderness of the part is connected with these chronically inflamed tissues, and not with the actual tubercular deposit. The persistence of this chronic inflammation implies the persistence of some cause of irritation, and without doubt the chief irritating cause lies in the tubercular tissue itself. The mere collections of cells forming the tubercles can of themselves hardly be irritating; it must be the chemical substances in the tubercle, the products either of the bacilli or of the cells themselves which keep up the process. Now there are many objections to Koch's view that the remedy acts by simple addition of substances to those already in the tissues, and it seems to me, for a variety of reasons which I cannot enter into now, that the material acts rather as a ferment, and for a time at least lessens or puts a stop to the production of these irritating chemical products by the bacilli or the cells. By this action the chief cause of the surrounding chronic inflammation is at once removed, and hence we have the rapid subsidence of this process. The microscopical evidence shows quite clearly that within three or four weeks there is very little disappearance of the actual tubercular tissue, that must evidently be a longer matter; while the clinical evidence shows diminution of swelling and pain, and in the case of matted glands, for example, subsidence of the periadenitis. I therefore believe that this diminished swelling and pain is in the first instance practically entirely due to cessation of the chronic inflammation around the tubercular area, and not to disappearance of tubercular tissue.

When this stage has been reached (and I am here speaking of cases treated by the intermittent method) matters seem in the majority of cases to come more or less to a

standstill, and the remaining swelling persists or only disappears very slowly ; indeed, in some cases the swelling again increases, but this early increase depends to a great extent on the manner in which the treatment is carried out. In only extremely few cases has the improvement gone on to apparently complete disappearance of the disease ; but it is not improbable, so far as I can judge from the cases I have had, that by means of the continuous method of administration the number of cases in which further improvement is obtained will be considerably greater. While the rapid improvement corresponds to the cessation of the chronic inflammation, the slow improvement corresponds to the removal of the tubercular tissue. Even where this is thoroughly carried out it must be a slow process, and the defect of the remedy seems to be that it somewhat rapidly loses its power, in the great majority of cases before the removal of the tubercular tissue has been completed.

In a few joint cases, after the preliminary improvement, progress has not only come to a standstill, but matters have got worse, in that suppuration has occurred. This took place in four cases, and I am under the impression with regard to two of them that the suppuration was possibly brought about by the treatment, by increasing the dose too rapidly, and thus keeping up too great local action.

We may therefore, I think, take it as a fact that in the great majority of suitable cases the early effect of the treatment will be to produce improvement, varying in amount according to the individual case and the mode of application of the remedy, and that subsequently this improvement will go on slowly or come more or less to a standstill. Further, it seems that the chances of complete cure by the remedy alone, within a few months at any rate, are slight, especially in cases where, as in unopened joints, the tubercular focus is buried in the tissues, and where, therefore, it cannot be got rid of except by the slow process of absorption.

In accordance with the various facts which have been mentioned we find that if the treatment is left off at the stage of improvement and cessation of reaction, or even within three and a half months (the limit of my experience on this point), recurrence will almost certainly take place. I have left off the treatment in ten cases which had more or less improved, after from two to nearly four months' treatment; and of these six have definitely relapsed, and in only one (No. 39) is there certainly no relapse. These cases are Nos. 15, 17, 22, 23, 24, 26, 32, 35, 38, and 39. The treatment has also been left off in a few other cases, but either there was no preliminary improvement or I do not know the result.

Such being the state of matters, there remains the question whether we can keep the disease at a standstill by going on with the treatment. (It may be noted in Koch's papers that he speaks almost entirely of a "standstill," and seldom of "cure.") This is an extremely important question, especially in cases where we cannot shorten matters by surgical measures. In a case of phthisis, for example, if we can bring the disease to a standstill and keep it so we have made a great stride in advance, and most patients would, I think, be willing and eager to go on with injections, even for an indefinite period, just as they winter in various health resorts with the same object in view, or as epileptics take bromide. Besides, an existing disease cannot really be at a standstill. If it is making absolutely no progress it is only a matter of time for it to be eradicated, or for the morbid tissue to become encapsuled; and if the bacilli are brought in contact with healthy tissues by removal of the tubercular elements, as histological examination shows to be the case where the tubercles lie in vascular tissue, they must die out in at most a few months, provided that they are not allowed to grow.

If now we examine the cases in my list which bear on this point (and my experience extends in a number of them over a period of nearly five months) we find that

the facts vary a good deal, due, no doubt, partly to the original distribution of the tubercular tissue, and partly to individual predisposition, more especially to the age of the patient; the result is apparently better in the young than in the old. In one case of lupus (No. 4), after two months' treatment the disease, which had not been entirely eradicated, was kept at a standstill for another two months by means of a decigramme once a week. Quite lately (since the abstract of cases was drawn up) this has seemed to be hardly sufficient, but a decigramme twice a week has checked it again. In one case (No. 11) a decigramme twice a week still keeps the disease in check, and in No. 12 improvement has steadily gone on up to the present time under decigramme doses given almost daily. In Nos. 16, 27, 28, 36, &c., similar doses given two and three times a week have also kept the disease from relapsing. In other cases, however, 2 (No. 6) and 3 (No. 3) decigrammes a week have failed to keep the disease in check, though the recurrences have been slow, and in No. 3 increase in frequency of the dose has again produced improvement. (The cases where scars have broken down after operations on joints do not come into the same category.)

On the whole I think we are warranted in saying that in a good many cases by keeping up and manipulating the injections with the view of maintaining the condition of standstill the disease can be kept in abeyance for five months, but that the frequency and strength of the injections necessary for this purpose vary in different cases. If the disease can be kept in abeyance for five months I see no reason why this condition should not be maintained for a longer time, and of course the longer the time the greater the chance that the tissues will destroy or encapsulate the remains of the disease. This action of the remedy is naturally of much greater importance in internal than in external tuberculosis. Physicians may be very thankful indeed, if they can do this, but surgeons cannot be satisfied in many cases with merely keeping the disease

at a standstill for an indefinite period; we have other means at hand by which we may hope to expedite the cure. Hence I have come to the conclusion that tuberculin as a main agent in treatment has more to do with the domain of the physician than with that of the surgeon.

It is clear from the above facts that the treatment does not come to an end, as was originally supposed, when the dose of one decigramme has been reached without causing reactions, but that if once the treatment is commenced it must be continued steadily and carefully; and so far as I can see we cannot as yet make any precise statement as to the period at which it may come to an end. This last fact is a very serious obstacle to its employment in many cases, but more especially in hospital practice, because, apart from the fact that patients soon become very irregular in their attendance, the expense of keeping up the treatment in anything like a considerable number of cases is very great; for example, the cost in tuberculin alone of keeping up my hospital cases just now is at least £1 a day.

Before going on to the last point I must say a few words as to the diagnostic property of the drug. A list of twelve cases will be found at the end of the abstract where injections have been made in doubtful and non-tubercular cases. The case of leprosy (No. 56) is a very interesting one, both as regards the local effects during the reactions and the statement of the patient that improvement has been obtained. Of the other cases the last three are important as showing fever without any definite local reaction. No. 57 is very interesting as showing the way in which the remedy reveals the existence of tubercular deposits. In this case I wanted to demonstrate the opinion which I had expressed that the knee trouble was not tubercular, and I therefore injected one centigramme. Next day I found that there had been fever. On inquiring further it was found that there had been no reaction in the knee, but the patient showed us two strumous scars on the arm which had reacted markedly. The facts as regards the diagnostic property of the fluid

seem to be that most non-tuberculous individuals do not show any symptoms with doses up to a centigramme, while, on the other hand, some suffer from fever without any local effect. On the contrary, the great majority of tubercular individuals react more or less violently, both generally and locally, with the same dose. Fever without local reaction does not necessarily imply tubercle; fever with local reaction almost always implies tubercle, with the exception of some cases of leprosy. Absence of reaction with a *first* dose of one centigramme almost absolutely excludes tubercle, but absence of reaction with a first dose of one or two milligrammes, or even after a dose of one centigramme, provided that it has been preceded by smaller doses, does not necessarily imply the non-tuberculous nature of the lesion.

Its Place in Surgery.

We now come to the last question which we have to consider, viz. what place should tuberculin occupy in the treatment of those forms of tubercular disease which come under the care of the surgeon?

Let us take first that form of tuberculosis in which the action of Koch's treatment is most manifest, viz. *lupus*. The experience of all who have treated cases of lupus by tuberculin alone has been that it is only very rarely indeed that it will produce a cure, and that we must go back to Koch's original statement that, in order to get a cure, operative measures must be combined with the treatment. Of my cases there is only one treated by tuberculin alone which seems to approach a cure, and it is of course too early as yet to speak positively. This is case No. 8, which is being treated by the continuous plan. On the other hand, the cases in which additional surgical measures are being employed promise exceedingly well. If we examine large sections of lupus we find that the tubercles are in parts aggregated together at the surface

of the skin, forming the visible nodules, and in other parts are scattered throughout the dense fibrous tissue of the skin or scar. By the ordinary methods of treatment, such as scraping and subsequent application of caustics—for example, strong nitric acid—the greater part of the visible disease is removed; but these isolated tubercles are left in places, and it is chiefly they which give rise to the recurrence. By Unna's salicylic plaster treatment, which yields fair results, the visible nodules are broken down more or less completely, and portions are removed which might very probably, be overlooked by scraping, but the isolated nodules are still for the most part untouched. By the combination of scraping and cauterisation, with subsequent use of these plasters after the wound has healed, a great deal is gained, but there is no security against recurrence on account of the isolated tubercles having escaped. Tuberculin, as we have seen from the histological facts, attacks and destroys these isolated tubercles pretty thoroughly, but is much less satisfactory in its action on the nodules. By combining the methods, however, we attack all parts, and everything seems to point to our being nearer a cure by this combination than by either plan separately. What I am now doing is this: first I give one, or better two, injections, fairly large so as to produce considerable local reaction. This brings into view all the nodules, and thus we are less likely to overlook them, while at the same time the tubercular tissue becomes softer and is more easily scraped away. I now scrape thoroughly and apply strong nitric acid, and then go on with the injections by the continuous plan, raising the dose qnickly, and aiming at cure by destruction of the tubercle rather than at standstill. In a few days the wound heals under the treatment, and then I go over the whole surface bit by bit with Unna's salicylic plaster method. In these cases I do not think that the injection treatment ought to be stopped in less than six months.

With regard to tubercular glands I must say that I have not seen any marked change in the glands themselves

as the result of this treatment. Some have somewhat diminished in size, but the great majority have remained stationary. No doubt this is often due to the fact that the glands are partly or wholly caseous, when of course no marked diminution can be observed. In one case (No. 38), where the glands were not only almost completely caseous, but where they were much matted together, and where their thorough removal would have been a matter of considerable difficulty, the periadenitis cleared up very markedly; and I think it is quite likely that in cases where there is much matting of the glands and tissues a short preliminary course of this treatment (about three weeks) would much facilitate their subsequent excision. Except for this purpose I do not think that it would be worth while carrying out the treatment in the case of tubercular glands.

In considering *tubercular diseases of bones and joints* I shall divide them into the two classes of aseptic and septic; including under "aseptic" those where the skin is unbroken or where chronic abscesses have been opened or operations performed and the wounds treated aseptically, and under "septic" cases those where there are suppurating sinuses.

Speaking first of those with unbroken skin and without chronic suppuration, we had twelve cases of disease of the larger joints, in two of which (Nos. 11 and 12) the signs of disease have, as far as I can see, completely disappeared; while in a third (No. 13) the result would, I believe, have been very similar but for the accident that the ligamentum teres was the main seat of disease, and had been destroyed by it. Whether this result is really cure, or only improvement with standstill of the disease, I cannot, of course, at present say, because the treatment is still being continued. In any case we have at present 16 per cent. with restoration of function, and possibly we might have had 25 per cent.; and the question is whether we should in the early stages of joint disease subject patients to this treatment on the chance that a result of

this kind may be obtained. Now, in the first place my cases are too few in number to justify us in drawing any conclusions as to the probable frequency of such a result. I feel no confidence that if I had had twenty-four instead of twelve cases I should have had any more in which this remarkable disappearance of the disease occurred ; while, on the other hand, I cannot say that under the continuous method of administration the results might not have been better. Besides, we must bear in mind that occasionally, though it is true very rarely, quite as complete restoration of the joint occurs under expectant treatment carefully carried out. I have had two or three such cases, though only after years of treatment, and usually in the best cases there is more or less limitation of movement. Again, suppuration has occurred since the treatment began in four of the cases, and in two of these I cannot but think that it was induced by the treatment, and it is not improbable that in some cases, if the treatment is pushed too fast, the increased local action may precipitate the impending suppuration. On the whole I think the probabilities are decidedly against cure by this treatment alone, although it is quite possible that if we could get a case in the very early stage, while only a few tubercles are present, and while they are still discrete, a cure might be accomplished. So far as I can see at present, the advice which I would give in cases which would previously have been treated by the expectant method is to treat them by that method, and not to employ the Koch treatment at this stage. I think, however, that this question is still open.

The next question is whether the treatment comes into play at a later stage, and, if so, when. For a time I thought that the place for this treatment would be in cases where expectant treatment had failed to arrest the disease, and before performing any operation. Employed at that period in the case I hoped, firstly, that a certain number of the cases might behave like Nos. 11 and 12, and that thus in these an operation might be avoided;

and secondly, that by the previous employment of the treatment the tubercular tissue might be so broken up that it could be easily removed by means of a comparatively trifling operation. This question I would still leave an open one, but my own inclination now is against the employment of the remedy at this stage for the following reasons. As regards the last point, we now know that it is not the tendency of this fluid to cause breaking down and softening of the tubercular tissue to any marked extent, and the hope that operations on joints would be more limited and more easily and thoroughly performed after this treatment has not been realised. As regards the first point, I think that the chance of avoiding operation at this stage is very slight, and certainly does not compensate for the drawbacks attaching to this course. The great objection to employing the treatment first is that tolerance has been established before operation, and that if recurrence takes place afterwards, and the scar breaks down, as happened in several of my cases, we no longer have this treatment to fall back upon ; and it is not at all remarkable that recurrence should take place in cases where only portions of the diseased tissues have been removed, for after the operation the joint contains lymph and blood-clot, which is some time in becoming vascularised ; and before vascularisation is complete tubercles may form in it, especially if any loose tubercular tissue has been left in the joint. For these reasons my own inclination is not to employ the remedy either in the early stage or before operation in joint diseases. It is possible, however, that one or two injections just before an operation might be of service in indicating the chief seats of the tubercular deposits, and in rendering them more easily removable.

It is at this period of the case, viz. after operation, that in my opinion the question of using tuberculin comes into play. Most of those who have employed the treatment have found that aseptic wounds or sinuses, or sinuses in which there is not much suppuration, heal as a rule very rapidly under it. In my list there are six cases of aseptic

sinuses, of which four have healed and the other two have improved, and one septic sinus in the soft parts, which was, however, only suppurating very little (No. 23), healed in twelve days. The position which I would at present assign to tuberculin in the treatment of disease of bones and joints is therefore as a means of obtaining healing of the wounds after operations have been performed. Where a complete arthrectomy or excision has been performed I would stitch up the wound, and wait till I saw whether healing occurred or not; if healing did not occur, and the appearance of the sinuses indicated recurrence of the tubercle, as indeed they usually do, and if they remained aseptic, I would employ tuberculin to get them to heal. Where the arthrectomy or excision was incomplete so far as the total removal of the tubercular disease is concerned, I would leave the wound open or provide very free drainage, and after about three weeks, when all the lymph on the surface had become vascularised, I would commence the treatment. Case No. 31 illustrates what I mean. It must always be remembered that unless the treatment is persevered in these sinuses will in all probability break down again, and I do not think it would be safe to leave it off in these cases either till the scar is well drawn in, or till at least six months have elapsed.

I think there is another possible advantage in tuberculin with regard to joints in that, by employing it after operation, it may not be necessary to do so much in the way of operation as might otherwise be required. I have always had a hankering after partial arthrectomies, but I have gradually been giving them up in favour of the complete operation; I think, however, that by the aid of tuberculin in getting the wounds to heal we may now in some cases be able to return to the less extensive operations, which in many ways present advantages over the other.

Where *septic sinuses* are present the treatment is for the most part contra-indicated, unless we can by operation render them aseptic previously. Under these circumstances I would follow the present lines of treatment in

these cases, and if the attempt to render the sinuses aseptic were successful, I would then take into consideration the question of Koch's treatment. In my list of six septic sinuses two cannot be said to have derived any benefit, and two were decidedly the worse for the treatment.

As to *genito-urinary tuberculosis* I cannot speak from my own experience, as I did not happen to have a case at the time I began the treatment, but I do not think that it will be applicable in most cases, for it is seldom that tuberculosis exists in the urinary tract for any length of time without becoming complicated with sepsis. At the same time, at the early stage before there is any marked septic complication, it seems to me that, considering the extremely serious nature of this form of tuberculosis, it would be quite right to give the treatment a trial. In that case one would not use the method so as to try to get a rapid cure, but rather with the view of getting and keeping up the condition of standstill. I have only tried it in one case of tuberculosis in this region, and that was in a patient of Mr. Rose's with tubercle in both testes, prostate, and various other parts of the body, to whom I gave a few injections. In this case there was no noticeable reaction in the testicles, but the amount of pus from the prostate diminished considerably.

I have had four cases of *tubercular laryngitis*, two of which healed and remained healed, and in one the treatment was given up as useless after a few injections (No. 48). In the fourth (No. 46) we committed the very grave error of giving up the treatment after a few injections because after the first there were no febrile reactions. When it was decided to resume the treatment we began at the dose we left off at, viz. 1 centigramme, and had a severe reaction, and subsequent extension of the ulceration. The treatment was given up again after ten injections, which had been administered at somewhat irregular intervals (latterly twice a week). I have since had serious doubts as to the wisdom of this second abandonment of the treatment, and I question whether the proper thing

would not have been to reduce the dose and pursue the continuous method.

As to *phthisis*, my opinion is not of course worth much, and I can only say that improvement has been the rule in the cases which I have had under treatment, as can be seen in the abstract of the cases. My own feeling is that if I had phthisis in a reasonably early stage I would have the treatment without any hesitation or delay. I would employ the continuous method with small but frequent doses, very slowly increased in amount, and aiming not at rapid cure, but at obtaining and maintaining a condition of arrest. With this object in view, I would not push the doses much above a decigramme, and I would from time to time diminish their frequency, the object being to have a certain reserve, so to speak, in hand, both as regards dose and frequency, so as to be able to combat any sign of recurrence by increasing the frequency and dose if necessary. Further, I should not look forward to leaving it off under from one to two years. In fact, I would try to make tuberculin occupy much the same position to phthisis that mercury does to secondary syphilis.

I have tried to bring forward as fairly and briefly as possible the facts which I have observed, and the conclusions which I would draw from them. These conclusions are, of course, only provisional, as the time has not yet come for dogmatising. That better results might have been shown I do not doubt, and with my present experience I believe that if I were going over the matter again I could show better cases. For in the first place we now know more about what cases are suitable and what are not, and we also understand more about the manipulation of the remedy, and can employ it with more success and less risk than formerly. Further, we must remember that this matter is still in its infancy, and I do not doubt that as time goes on improvements will be made. The great failing of the material in its present form is the rapidity with which the tissues become tolerant of it; and it is to be hoped that when the question of

tolerance has been more studied, some way will be devised of preventing it. Besides, the fluid as we obtain it contains not only the active principle, but also other substances, which, as has been found in the case of other drugs, may more or less hamper its action; and thus, when the attempts to separate the active agent in a pure state have proved successful, we may find that some of its present disadvantages have been got rid of. Even in its present form it must take its place among our means of combating tuberculous disease, although without doubt, in surgery, that place must be a secondary one.

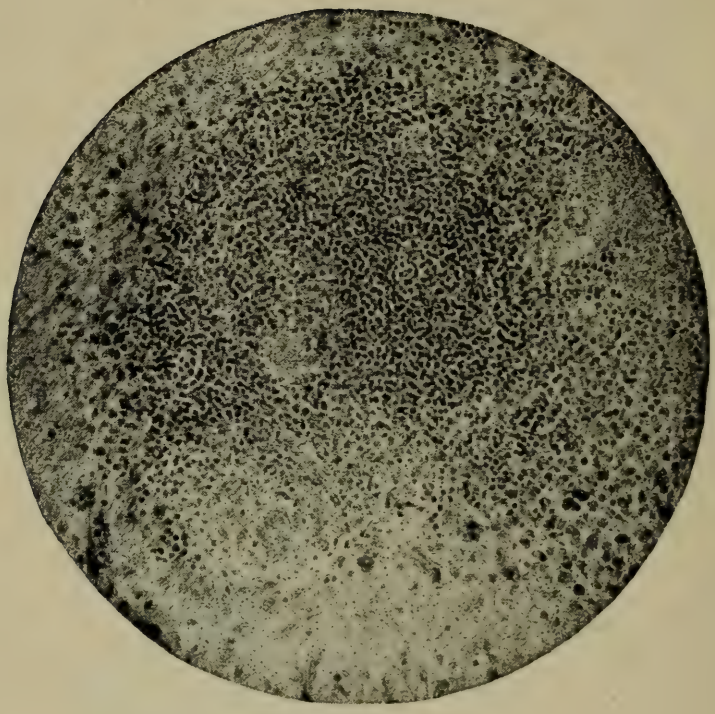

Fig. 1.

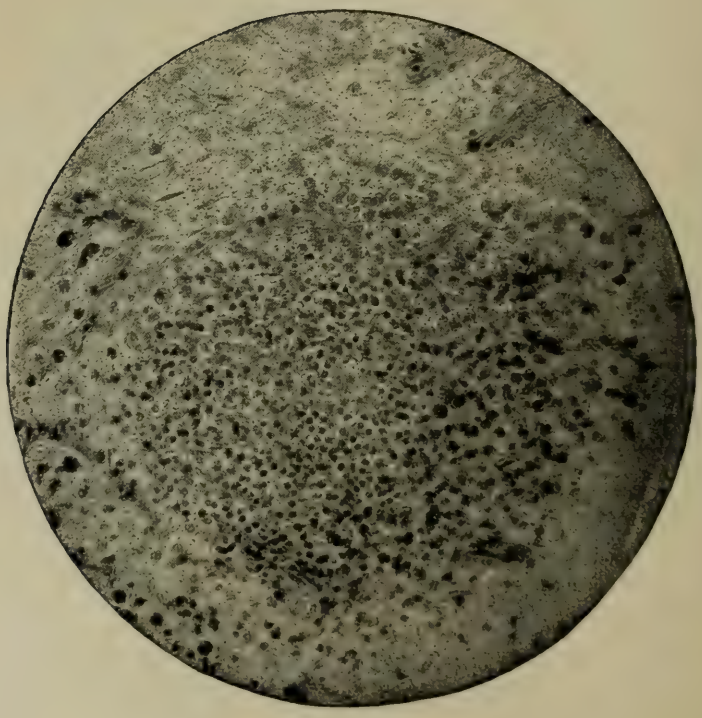

Fig. 2.

DESCRIPTION OF PLATE III.

These illustrations are from photographs taken by Mr. Andrew Pringle from sections (chiefly of synovial membrane), from cases which had been treated by tuberculin. The method of reproduction is not a suitable one, and does not in any way do justice to the photographs; if, however, the plates are looked at from a distance they show in a general way the appearances which were present.

FIG. 1.—Section of synovial membrane showing small-celled infiltration of a tubercle: towards the centre some remains of epithelioid cells can still be found.

FIG. 2.—A similar appearance. In addition, larger cells are seen outside the small-celled infiltration.

DESCRIPTION OF PLATE IV.

FIG. 3.—Two tubercles with commencing fibrous formation around the giant-cells.

FIG. 4.—Formation of young fibrous tissue in a tubercle; a few epithelioid cells were still present at each side.

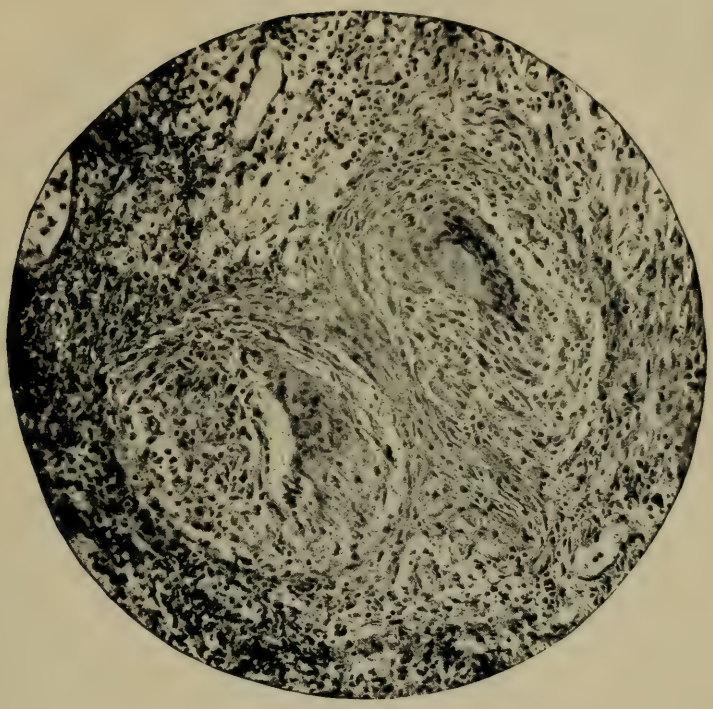

Fig. 3.

Fig. 4.

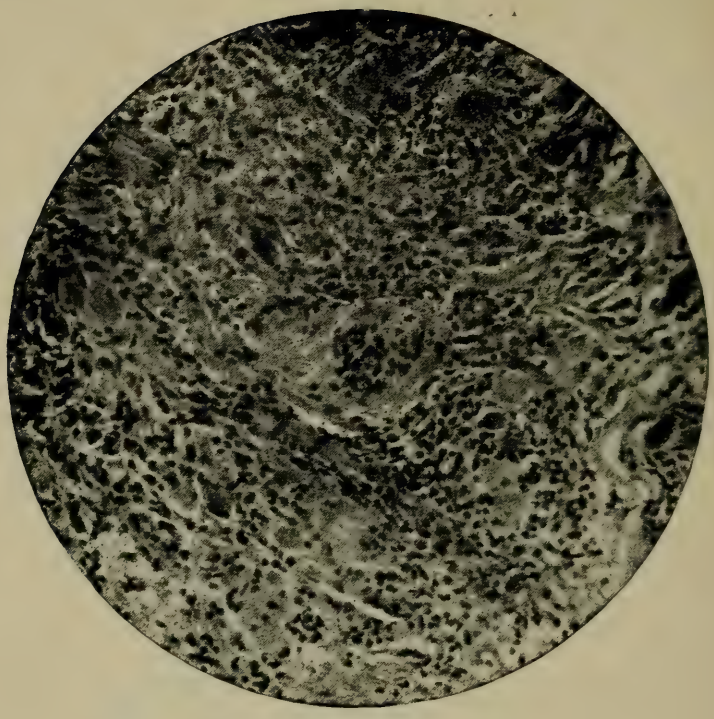

Fig. 5.

Fig. 6.

DESCRIPTION OF PLATE V.

FIG. 5.—Section from a case of lupus after two and a half months' "continuous" treatment, showing a giant-cell still present among the young fibrous tissue.

FIG. 6.—Section of synovial membrane under a low power. Two tubercles still present with marked small-cell infiltration. Much infiltration also towards the free surface, but no distinct remains of tubercles.

ABSTRACT

OF ALL

CASES OF TUBERCULAR DISEASE

WHICH HAVE BEEN TREATED

BY MR. WATSON CHEYNE

WITH TUBERCULIN

UP TO THE BEGINNING OF APRIL, 1891.

THE amount administered at each injection is stated, and an attempt has been made to indicate the occurrence and intensity of the general reactions, and the frequency with which the injections were given. The strength of the reactions is indicated by dashes placed above the dose. Where there is no dash there was no reaction. Where there is one dash (*e.g.* ·002′) there was only slight reaction, the temperature perhaps reaching 100° or a little higher, but without the patient suffering to any noticeable extent, and often not at all. Where there are two dashes (*e.g.* ·002″) there was moderate reaction, the temperature being usually above 101°, between that and 104°, and the various symptoms being moderate in intensity. Where there are three dashes (*e.g.* ·002‴) there was a severe reaction, that is to say, the temperature was usually at or above 104°, though sometimes it was only 103°, and the general symptoms were correspondingly severe. Where

there are four dashes (*e. g.* ·002″″) the condition of the patient was such as to make us anxious; only two reactions of this kind will be found in the list. No attempt has been made to indicate the local reaction.

This method, of course, only roughly indicates the state of matters. In some cases, even where the temperature was up to 102°, the patients did not suffer at all, but these I must mark as moderate reactions. In other cases patients with a temperature of 102° or 103° (within my "moderate" area) have suffered more than those with a temperature of 104°. Where this has been the case I have indicated them as severe. Those with slight reaction, *i. e.* with elevation of temperature above 99°, but seldom with any other symptoms, are probably correct. Those marked severe are, no doubt, all correct. But among those marked "moderate" are some where the symptoms were very slight, and which, were it not for the temperature, ought to have been so indicated, and others which were on the border-line as regards symptoms between moderate and severe, but where the temperature was not particularly high. The idea of indicating reaction and frequency in this way was taken from a paper by Leichtenstern, but his plan has been somewhat modified.

The frequency of the reactions is also indicated in the following manner:—Where more than one injection was administered on the same day the doses are bracketed together with a comma between them, thus: (·002, ·002). Where the injections were given on succeeding days there is a hyphen between each dose, thus: ·002–·002. Where there was one clear day's interval between the injections it is written thus: ·002 ; $_1$·002; where there was two days' interval thus: ·002 ; $_2$002, and so on.

As the ultimate result depends to a great degree on the way in which the material is used, it is absolutely essential in publishing the records of cases that the above facts should be clearly indicated, otherwise it is impossible to judge of the value of the results.

It is, of course, difficult in an abstract to convey a

thorough idea of any particular case, and I have attempted to add to the value of the record by stating, in the first place, what I would have done in the case in question before the Koch period, and in the second place, what I would now do in a similar case.

K. C. H. = King's College Hospital.

P. G. C. H. = Paddington Green Children's Hospital.

I.—Cas

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
1	N. L., æt. 20, female, K. C. H., admitted Nov. 25th, 1890; discharged Dec. 30th, 1890 *Diagnosis.*—Lupus of neck and arms; disease of metacarpal bone	One uncle died of phthisis. About 12 years ago had tubercular glands in neck which suppurated. The wounds did not heal and lupus appeared around them, and spread along each side of neck. Since that time abscesses have formed on the inner side of both arms, and patches of lupus have also appeared. About 5 years ago the metacarpal bone of the little finger of her right hand began to swell, and portions of bone have been removed. She has undergone many operations and much treatment	Internal organs healthy; there is one large cicatrix around the under part of the jaw, and a number of smaller ones lower down. Numerous lupus nodules around the upper border of this scar, extending on to the cheek and occupying a breadth of about an inch, covered with crusts. Parts of the cicatrices are apparently healthy, but everywhere one sees lupus nodules here and there scattered over them. There are a number of patches covered with crusts along the inner side of both arms. The metacarpal bone of the right little finger is much thickened and very tender, and there is a small sinus in front, which she says has existed for 4 years	·01‴; $_{4}$·01‴; $_{16}$·002‴ (patient declined further treatment)
	Re-admitted Jan. 26th, 1891	—	On re-admission a few fresh lupus nodules were found around the edge of the scar in various parts, and the finger, which was painless and not swollen when she was discharged, was as bad as it was originally; the sinus had opened up again and a fresh one had formed. Portions of the scars on the arm were red, and here and there lupus nodules were seen	·001″; $_{1}$·001–·001–·001–(·001,·001′)–(·001,·001)–·002′(·002, ·002)–(·002, ·002)–(·002′, ·002)–(·002, ·002)–(·002, ·002)–(·002, ·002)–(·002, ·002)–(·002, ·002)–(·002, ·004)–(·004, ·004, ·004)(·004, ·004, ·004)–(·004, ·006)–(·006, ·006, ·006)–(·008, ·01)–(·01, ·01)–(·02,·02)–(·03, ·03)–(·04, ·04)–(·05′, ·05)–(·05′, ·05′)–(·06, ·06)–(·07,·08)–(·09, ·1)–(·1, ·1)–·1; $_{1}$(·1, ·1)–·1–(·1, ·1, ·1″); $_{1}$(·1, ·1). Began an out-patient on March 5th to go on with ·12 every day
2	Miss D., æt. 42, under the care of Dr. Thin. *Diagnosis.*—Lupus of face	Tubercular family history. Disease began 25 years ago as a spot in the middle of the cheek. Has been treated in all sorts of ways, as the disease has been steadily progressing, especially towards the eye. We have lately been considering the advisability of excising the whole patch, either at once or in portions, and closing the wound immediately by Thiersch's skin grafts	Internal organs healthy. On the right cheek there is an extensive patch of lupus extending as far out as the centre of the malar bone, as low down as the level of the angle of the mouth, and as high up as the middle of the lower eyelid, and extending along the inner side of the nose as high as and quite close to the inner canthus of the eye. On the cheek the lupus nodules are embedded in dense scar tissue, and much of it is ulcerating and covered with scabs. There is a scar on one buttock, which the patient says was a lupus patch, which commenced when she was 6 years old, and which spontaneously disappeared after several attacks of acute inflammation in it	·006″; $_{3}$·006‴; $_{4}$·007″ $_{6}$·008″; $_{8}$·005″; $_{10}$·005″ $_{7}$·003′; $_{2}$·003′; $_{1}$·003″; $_{1}$·002 $_{1}$·003; $_{1}$·003; $_{2}$·006′; $_{1}$·008′ $_{1}$01; $_{2}$·01; $_{1}$·012; $_{1}$·015 $_{2}$·02; $_{1}$·03; $_{1}$·05; $_{1}$·07; $_{1}$·1 $_{1}$·13; $_{1}$·15; $_{1}$·2; $_{1}$·27; $_{1}$·32 $_{1}$·4; $_{2}$·45; $_{1}$·4; $_{3}$·2; $_{1}$·2 $_{1}$·25; $_{1}$·3; $_{1}$·33; $_{1}$·35; $_{1}$·35 $_{1}$·35; $_{1}$·38; $_{1}$·35; $_{1}$·38; $_{1}$·4 $_{1}$·4; $_{1}$·3–·25–·3–·3–·3–·35–·35 To continue. Stopped in May. Very rapid recurrence necessitating operative interference

OF LUPUS.

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Nov. 26th, 1890; 3rd injection on Dec. 17th, 1890; discharged at her own request on Dec. 30th, 1890	None	When discharged on Dec. 30th the lupus patches had entirely disappeared, the sinus on the finger had healed and the swelling and tenderness of the bone had gone	The first reaction was a severe one but there were no dangerous symptoms; much local reaction. The temperature reached 104·4°, 16 hours after the injection fell a little, and again 31 hours after the injection was 104·8°. It then fell slowly, but was still about 100° when the second injection was given on Dec. 1st. There was an erythematous rash over the chest. The reaction after the second injection was much more severe, and for a few hours I was rather anxious about her. The temperature reached its highest point (105·2°) 9 hours after the injection, and during the next 24 hours she was retching a good deal, coughing, had a very weak and rapid pulse (168) and rapid respiration (60). She was also partially unconscious. Stimulants were administered and the symptoms gradually passed off, but the temperature did not reach normal till 7 days after the injection. There was violent local reaction and the lupus nodules for the most part sloughed out. There was a slight trace of albumen on Dec. 4th, but this disappeared next day. After the 3rd injection, though the temperature reached 104·4°, the general effects were comparatively slight.
Injections recommenced on Jan. 28th. Total number of injections up to April 8th = 100. Total amount of iodid used = about 5·816 grms.	On March 15th Unna's strong salicylic and creosote plasters were applied over the remains of the lupus on the right side of the neck	Note on April 6th.—"Some spots of lupus can still be recognised on the neck, but the condition is steadily improving. The arms are very much better. The sinuses on the hand are still unhealed, but the swelling is less and free from pain or tenderness. On the right side of the neck are some sores due to the salicylic plasters" *Result.*—Very considerable improvement; still progressing	The patient did not suffer during this course of treatment. The parts around the lupus patches were kept constantly red and scaling, and these patches have gradually become less visible. The hand also steadily improved, and the swelling and tenderness disappeared. On March 15th remains of the lupus nodules were still visible in places in the neck and salicylic plasters were ordered for these places; the patches on the arm were very much improved and the finger was painless, but the most recent sores had no t yet quite healed. She complained a good dea lof her hair falling out during the course of the treatment. Treatment stopped in the middle of May. Slight recurrence in July.
Commenced Nov. 27th, 1890. Total number of injections up to April 8th = 51. Total amount of iodid used up to date = 8·095 grms.	From time to time remnants of lupus tissue were picked out with a fine spoon, and on Feb. 16th the use of Unna's salicylic plasters was begun	Note on April 8th.—"The cheek is still in parts ulcerated as the result of the plasters, but the part looks quite healthy. Till this heals one cannot of course say how much has been gained" *Result.*—Improving	The patient suffered a good deal after the early injections, there being sickness, headache, weak pulse, and pains, at first in the back, and subsequently in the limbs. After the first 3 or 4 injections she complained that at the height of reaction her limbs were powerless, and the pain in them so great that she could not move. As it seemed not improbable that there was some hysteria present, we gave an injection of the carbolic lotion without tuberculine, but no symptoms followed. By persevering steadily and

utiously these troubles were gradually overcome, and we were enabled to raise the dose. She also had veral skin eruptions, mainly erythematous. On one or two occasions several subcutaneous hæmorrhages curred on the thighs after the injections. On March 2nd, after the injection of 4 decigrammes, she mplained in about 10 minutes of great depression and of the sudden appearance of urticaria over the whole dy. These symptoms very soon passed off. She had a second attack of urticaria a few minutes after the jection of ·38 grm. on March 22nd, but without any marked depression. The lupus steadily improved, ough at first more slowly than usual, owing no doubt to the intervals between the injections and the small dose. there were evidently still some superficial remnants of lupus tissue the treatment by salicylic plasters was gun on Feb. 16th, a treatment which she had gone through several times before without any permanent benefit.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
3	M. C., æt. 26, female, admitted Dec. 1st, 1890, K. C. H. *Diagnosis.*—Lupus of face and palate; enlarged glands in neck	Mother died of phthisis. Disease began 15 years ago. The nose was first attacked, and from thence it spread on to both cheeks. Hard palate and upper gums became affected about 5 years ago and the upper lip 2 years ago. Has undergone all sorts of treatment. Scraped last on Nov. 21st, 1890	Internal organs healthy. Very extensive lupus. A considerable part of the cartilaginous part of the nose has been eaten away as well as the columna. It extends up over the root of the nose, and for a short distance on to the forehead. From the nose it extends on to both cheeks, on the right side covering the whole of the cheek down to the lower border of the inferior maxilla, and as far outwards as the level of the anterior border of the masseter. On the left side the disease does not extend so low, hardly reaching below the level of the angle of the mouth. The upper lip is much affected and swollen, and the disease spreads to the inner side of the lip, from thence to the front of the gums; there was also a small patch at the anterior part of the hard palate on the right side. Much of the surface is raw as the result of the recent operation. There was some discharge from the nose. There are some enlarged glands in the submaxillary region and at the angle of the jaw on the right side	·006‴; $_{2}$·006″; $_{1}$·006″; $_{1}$·008″; $_{1}$·008″; $_{1}$·008″; $_{2}$·01′; $_{1}$·01; $_{1}$·013; $_{1}$·02; $_{4}$·03′; $_{2}$·06; $_{1}$·06′; $_{1}$·08′; $_{2}$·1′; $_{3}$·1′; $_{2}$·1; $_{1}$·1; $_{1}$·1; $_{2}$·12′; $_{1}$·12; $_{1}$·12; $_{2}$·12′; $_{1}$·12; $_{1}$·15; $_{2}$·12; $_{1}$·12; $_{1}$·12; $_{1}$·12; $_{2}$·12; $_{2}$·1; $_{1}$·1; $_{2}$·1; $_{1}$·1; $_{1}$·1; $_{2}$·1; $_{1}$·09; $_{1}$·1; $_{2}$·1; $_{1}$·1; $_{1}$·1; $_{2}$·1; $_{1}$·1; $_{1}$(·1,·1)–·1–(·1,·1)–·1–·1–·1–(·1, ·1)–(·1, ·1)–(·1, ·1)–(·1, ·1, ·1)–(·1, ·1, ·1)–(·1, ·1, ·1)–(·1, ·1, ·1)–(·1, ·1, ·1)–(·1, ·1, ·1)–·12–(·12, ·12)–(·12, ·12, ·12)–(·12, ·12, ·12)–(·12, ·12, ·12). Treatment being continued
4	R. S., æt. 25, female, admitted Nov. 29th, 1890, K. C. H.; discharged Feb. 2nd, 1891, to attend as out-patient *Diagnosis.*—Lupus of nose and cheek	Father and mother consumptive. Began on upper lip, immediately below septum of nose, 5 years ago. Has steadily extended in spite of all sorts of treatment. Has had 24 operations, the last about 6 weeks before admission	Various organs healthy. About ⅓rd of the cartilages at the tip of the nose have been destroyed, especially on the left side; the orifices of both nostrils are considerably contracted. The centre of the upper lip is drawn up. The greater part of the nose is covered with scar tissue, in which are numerous lupus nodules and small ulcers. On the left side there is a separate patch of lupus close to the inner canthus of the eye. There is also a patch about the middle of the right cheek, which is ulcerated, and below the symphysis of the jaw is a thickened scar about 1½ in. long, probably from a suppurating gland. There was evidently some disease inside the nose, but the orifice on the left side was so small that nothing could be seen	·008‴; $_{2}$·01″; $_{3}$·01″; $_{1}$·01″; $_{1}$·01″; $_{2}$·01″; $_{1}$·01′; $_{1}$·013′; $_{1}$·02′; $_{3}$·03′; $_{2}$0·4′; $_{1}$·06′; $_{1}$·08′; $_{2}$·1′; $_{3}$·12′; $_{2}$·12; $_{3}$·15; $_{2}$·2″; $_{3}$·23′; $_{2}$·1; $_{4}$·1; and once a week ·1
5	H. F., æt. 15, male, admitted Dec. 1st, 1890, K. C. H.; discharged Jan. 28th, 1891. *Diagnosis.*—Lupus of thigh and axilla	No phthisical family history. Disease began 6 years ago	Internal organs healthy. At the back of the right thigh, rather below the middle, is a patch of lupus 3½ in. from above downwards and 3½ in. transversely. The patch is much thickened and covered with scales; there is no ulceration, and no signs of cicatrisation. Towards the outer part of the left axilla there is a similar patch, 3½ in. from above downwards, and 2½ in. from side to side	·01‴; $_{2}$·01″–·01″; $_{2}$·013″; $_{1}$·013″; $_{1}$·015″; $_{2}$·02′; $_{1}$·022′; $_{1}$·024′; $_{1}$·03′; $_{4}$·04′; $_{2}$·05′; $_{1}$·06; $_{1}$·08; $_{2}$·1: $_{3}$·12′; $_{2}$·1; $_{3}$·12; $_{2}$·12; $_{3}$·1; $_{2}$·1. Subsequently attended twice a week and received ·1. Not been seen since the beginning of March

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Dec. 2nd, 1890. Number of injections up to April 8th, 1891 = 93. Total amount of fluid used up to April 8th, 1891 = 8·705 grms.	Sent to convalescent home on Feb. 15th for a month. Readmitted on March 16th. On March 20th a number of nodules on the left cheek were scraped out and cauterised with nitrate of silver. On April 3rd the right side of the cheek was treated in a similar manner. In portions excised on March 20th there was still a good deal of tubercular tissue present	On April 8th the left cheek is still scaling in places, but there is no fresh recurrence; the parts scraped have healed. On the right side there are several sores, the result of the previous scraping *Result.*—Great improvement in the lupus for a time; relapse. Subsequent improvement. Glands in *statu quo*	Nothing abnormal about the reactions. The patient complained chiefly of headache, slight cough, sore throat, and the discomfort of the local reaction over this wide area. There was a good deal of local reaction, and after the first week the part steadily improved (though more slowly latterly) till she was sent to the convalescent home on Feb. 15th. On the morning of the previous day it was noticed that there were several small quite superficial bodies, almost like vesicles, on the skin just outside the lower angle of the lupus patch on the right side (on microscopical examination these were found to be really vesicles). Since the beginning of March a number of nodules have appeared at several parts of the scar, more especially towards the edge, and when readmitted on March 16th there was a considerable number of nodules over the surface, and ulceration was occurring at the nose. The left cheek was operated on as above, and the frequency of the injections was increased. Considerable improvement followed the increased frequency, and a large number of the nodules faded away. As there was still some tendency to ulceration on the nose and at one part of the cheek, these points were scraped and touched with nitrate of silver on April 3rd. Treatment stopped at the end of May, then much improved.
Commenced Dec. 3rd, 1890. Total number of injections up to April 3rd, 1891, = 30. Total amount of fluid used up to April 3rd, 1891, = 2·331 grms.	None	Note on April 3rd.—"The parts remain healed, and there is no tendency to relapse. There are still several yellow spots in the scar, and the lupus patches below the eye and at the angle of the nose are not quite level." *Result.*—Great improvement. As yet no relapse	Before the temperature rose, after the first injection, patient had a rigor. She also complained of sore throat, nausea, and headache. There was a trace of albumen 2 days later, but this did not recur. The sore throat recurred after the first 9 injections; there was redness of the fauces, but no ulceration. No other noteworthy symptoms. The lupus improved rapidly and very markedly. Treatment stopped in the middle of May. Lupus nodules still present.
Commenced Dec. 3rd, 1890. Total number of injections = about 30. Total amount of fluid used = about 2·057 grms.	None	"When last seen, about the beginning of March, there were still remnants of lupus in both places, but most on the leg. The disease seemed to be quite stationary." Patient has not been seen since, and cannot be found. *Result.*—Considerable improvement, and then standstill	After the first injection patient had a papular rash in patches over his back and sides, and slightly over the chest and thighs, in position and general appearance not unlike a herpes zoster. This faded away and disappeared about Dec. 14th. On Dec. 22nd a small abscess was found, and opened at anterior fold of axilla, and rapidly healed. The local reaction was fairly severe, but the patient was not very ill. Tubercles were still present in tissue removed from the thigh in the middle of January, though evidently much atrophied and destroyed.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
6	E. D., æt. 60, female, admitted Dec. 6th, 1890, K. C. H.; discharged on Feb. 12th, 1891, to attend as out-patient. *Diagnosis.*—Lupus of face	No phthisical family history. Disease began about 8 years ago on the outer side of the right nostril, and has spread very slowly. Patch over left malar bone began 4 years ago. Is sure that there was no lupus in early life. No treatment	Internal organs healthy. Heart sounds normal, but beat irregular and intermittent. There is a red, and in places ulcerated, patch over the lower $\frac{3}{4}$th of the nose. This is continuous with an oval patch over the right malar bone and cheek. On the left side there is a patch over the left malar bone and cheek, almost symmetrical with that on the other side, but not continuous with the disease on the nose. Both of these patches are in parts ulcerated and covered with crusts. The whole of the upper lip is thickened and red, with nodules scattered over it. The mucous membrane on the inner surface of the upper lip is ulcerated	·01′′; $_1$·01′; $_1$·015′′; $_1$·02′′; $_2$·02′; $_1$·02′; $_1$·023; $_1$·03; $_4$·04′; $_1$·05; $_1$·07; $_1$·1; $_1$·12 $_2$·15; $_3$·1; $_2$·1; $_1$·1; $_1$·12; $_2$·12; $_1$·1; $_1$·12; $_2$·12′; $_1$·12′; $_1$·1; $_2$·12; $_1$·12; $_1$·1. Subsequently attended twice a week and received at first ·1, and after March 11th ·15
7	S. T., æt. 40, female, nurse at the North-Western Hospital *Diagnosis.*—Lupus of face and neck	When 3 years old burned right cheek below ear. That never healed up, but did not extend till she was about 18 when it began to spread downwards under the jaw. It has steadily spread since and for about 12 years has been extending over the cheeks. Been operated on many times, used Unna's salicylic plasters and a variety of other treatments; has never healed up	Internal organs healthy. Extensive lupus involving the greater part of the right side of the neck in front, extending under the jaw to the left side. Spreading on the left cheek as high as the lower eyelid, forward to side of nose and near angle of mouth, and backward behind the ear. On the right cheek it is not quite so extensive as on the left side and there is a good deal of apparently healthy scar tissue. The whole surface except the scar tissue on the right side was covered with lupus nodules which were ulcerating and covered with scabs	·006′′′; $_4$·006′′; $_3$·006′; $_2$·006; $_4$·006′′; $_2$·005′′; $_2$·006′′ (week's interval); ·006′; $_1$·008′; $_4$·01′; $_1$·015; $_1$·02; $_2$·04; $_1$·06′; $_1$·08; $_1$·1; $_4$·1; $_6$·1; $_2$·1; $_3$·1; $_2$·1; $_3$·1; $_6$·1; $_2$·1; $_2$·12; $_3$·15; $_1$·15; $_1$·15; $_2$·15; $_1$·15; $_2$·15; $_1$·15; $_2$·15; $_1$·15; $_1$·15. Treatment stopped
8	F. N., æt. 17, male, K. C. H.; admitted Jan. 14th, 1891 *Diagnosis.*—Lupus of face and neck; enlarged glands in neck	No tubercular family history. Has suffered from lupus for 14 years, and has been operated on many times at various skin and general hospitals. Began on left cheek, and during the last year has extended to the nose and upper lip	Internal organs healthy. The whole of the left cheek is affected with lupus, being composed of scar tissue with numerous lupus nodules, many of them undergoing ulceration. The left ear is much destroyed and there is active ulceration going on there. The left upper eyelid is also covered with ulcerating lupus patches and there is very marked ectropion on the left side. The left cornea is opaque. The tip of the nose is also ulcerating and the disease has destroyed some tissue there, affects the columna and extends a short distance into the interior. The whole of the upper lip is affected. The lupus extends below the jaw on the left side over and under the chin and slightly on to the right cheek. There are several enlarged glands in the neck	·002′′; $_1$·002′′; $_2$·002′′; $_1$·002′′; $_1$·002′′; $_2$·002′–·002–·002–·002–(·002, ·002)–(·002, ·002)–(·002, ·002)–(·002, ·002, ·002)–(·002, ·002, ·002)–(·002, ·002, ·002)–(·002, ·002, ·002)–(·002, ·002′, ·002)–(·004, ·004′)–(·004, ·004)–(·004, ·004, ·004)–(·004, ·004)–(·006, ·006)–(·006, ·006)–(·006, ·006)–(·006, ·006)–(008, ·008)–(·008, ·008, ·008)–(·01, ·01, ·01); ($_1$·015, ·015)–(·02, ·02)–(·03, ·03)–(·04, ·04)–(·05, ·05)–(·06, ·06)–(·07, ·07)–(·08, ·09)–(·09, ·1)–·1; $_1$·1, ·1)–(·1, ·1)–(·1, ·1)–(·1, ·1)–(·1, ·1)–(·1, ·1)–(·1, ·12)–(·12, ·12)–(·12, ·12)–(·12, ·15)–(·15, 15). Since this date (March 15) he has continued the injections twice daily, gradually increasing the dose up to 2 decigrams. From the 2nd of April this dose was given thrice daily

Total number, mount, &c., of injections.	Additional treatment.	Condition when last seen	Notes.
Commenced ec. 8th, 1890. tal number of jections up to pril 6th, 1891, = 41. tal amount of id used up to that date = 3·818 grms.	On March 20th the strong salicylic plasters were applied to the left cheek	Note on April 6th.—"The condition of the face is better than when the patient was admitted, but shows a distinct tendency to relapse. On the left cheek the lupus nodules are breaking down under the plaster" *Result.*—Improvement for a time, but tendency to relapse. Treatment stopped in May. Recurrence	The general constitutional disturbance after the reactions was not marked. There was a slight erythematous rash on the chest after the first injection. The local reaction was not so marked as in other cases, but was quite distinct. When she was sent home there were very few nodules remaining, but these have somewhat increased in number. On March 20th the strong salicylic plasters were applied to the left cheek.
Commenced c. 10th, 1890. of injections to April 8th, 1891 = 35. tal amount of id used up to ril 8th, 1891 2·780 grms.	Patient was advised to apply Unna's salicylic plasters to one side, but she did not get the proper material	Note on April 5th, 1891.—"The condition of the lupus is markedly better than before the treatment wascommenced, but there is still lupus tissue round the greater part of the margin and at one or two places in the scar. The appearance is not so good as it was some weeks ago" *Result.*—Considerably improved, but tendency to relapse	After the first injections patient had a scarlatiniform rash over the trunk. The lupus steadily improved for some weeks till considerable intervals were left between the injections, when it began to show signs of going back. Lately the injections have been given more regularly and frequently, and the condition has remained pretty stationary. Patient was ordered to apply Unna's salicylic plasters to the margin of the scar about the middle of March, but did not carry out her instructions properly.
ommenced n. 16th, 1891. al number of ctions up to ril 8th, 1891 = 147. al amount of d used up to that date 13·246 grms.	Boracic ointment to prevent scabbing	Note on April 8th—"There is everywhere a smooth depressed cicatrix which is gradually becoming paler. No trace of lupus tissue. Glands in neck rather smaller" *Result.*—Very remarkable improvement. Disappearance of lupus tissue. Glands smaller	There was no serious general disturbance, but the local reaction was marked, and for several weeks the seat of the disease was red and scaling. The last places to cease scaling were the upper eyelid, the tip of the nose, and the left ear, and these had ceased to scale at the end of March. In a piece excised from the margin at the middle of March there were only a few traces of tubercular tissue. In a piece removed at the beginning of April no lupus tissue was found. Treatment stopped in the middle of May. No recurrence in July. Some strumous glands in neck suppurated at the end of May and were excised.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
9	Miss C., æt. 43, came under treatment on Feb. 28th, 1891. *Diagnosis.*—Lupus of face, enlarged glands in neck	Tubercular family history. The disease began 20 years ago in the middle of left cheek, and has only attacked the nose during the last 2 years. Has undergone a variety of methods of treatment	Internal organs healthy. A number of enlarged glands in neck, some of which have suppurated. The whole of the left cheek is the seat of lupus, which extends on to the left ear, part of which is destroyed, and down into the neck and under the chin to the right side. The left upper eyelid is also extensively affected, and there is marked ectropion of the lower lid. It also extends on to and affects the whole of the nose, the part over the cartilage being one mass of lupus tissue. The corresponding part of the interior of the nose is affected, and there are several spots on the upper lip. There is also an extensive patch of ulcerating lupus over the right cheek. On Feb. 20th I scraped the lupus as thoroughly as possible, applying nitric acid to the raw surfaces. There were, however, numerous nodules which I could not remove, as they were embedded in dense scar tissue, and I only scraped the nose superficially, for if I had attempted to do it thoroughly the whole of the cartilaginous portion must have come away	·002″, $_2$·004″; $_1$·007″; $_1$·01″; $_1$·01′–·01′–·01′–·01′–·01″–·01′–(·01, ·01″); $_1$·01′–·01′–(·01′, ·01)–·012″–·012′–(·012, ·012)–·015–(·015′, ·015)–(·015′, ·015)–·02″–(·01′, ·01)–(·01′, ·01)–(·01′, ·01)–(·013′, ·01–(·01′, ·01)–(·012′, ·015)–((·015, ·015)–·02–(·02′, ·015)–(·02, ·02)–(·02′, ·025)–(·025′, ·03)–(·03, ·035)–(·035, ·04)–(·04, ·05). To continue
10	W. H., æt. 21, male, K. C. H.; admitted Feb. 23rd, 1891. *Diagnosis.*—Lupus of face, hands, and leg, enlarged glands in neck	No tubercular family history. The lupus on the face began 11 years ago, commencing on the left cheek, and has been spreading steadily ever since, in spite of various operations and other treatment. When 6 years of age had tubercular disease of one toe; this was followed 2 years later by disease of right elbow and of upper part of right tibia	Internal organs healthy. Enlarged glands in neck. Extensive lupus of the face, involving both cheeks, upper lip, and the whole of the nose, a portion of which has been destroyed, and extending on both sides below the jaw, meeting at the chin. There is a large patch of lupus on the back of the left hand, and a smaller one on the back of the left forearm at the lower part. Sore on left leg, which he states was the result of an injury a month before admission. On Feb. 25th, before commencing the Koch treatment, the left side of the face was well scraped, but the nose and the right side were left untouched	·002‴; $_1$·002″; $_1$·002‴; $_2$·002‴; $_1$·002‴; $_1$·001″; $_1$·001″–·001″; $_1$·001–·002″–·002″–·002′; $_1$·003′–·004″–·005′–·005′–(·005, ·005)–·006′–(·006, ·006)–·006–(·006, ·006)–(·006, ·006)–(·006, ·006)–(·008, ·008)–(·01′, ·01, ·01)–(·01, ·01, ·01)–(·02, ·02, ·02)–(·02, ·02, ·02)–(·03, ·03, ·03)

Total number, nount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced eb. 25th, 1891. tal number of jections up to pril 8th = 53. tal amount of uid used up to that date = ·881 grms.	Boracic ointment. Unna's strong salicylic plasters were commenced on March 12th	Note on April 8th, 1891.—"The condition of the face is very greatly improved. Parts are broken down under the influence of the salicylic plasters: portions where these have been applied are now soundly healed, and at present show no trace of disease. The parts to which the plasters have not yet been applied are still rapidly improving. Glands smaller" *Result.*—Improving steadily	The patient did not suffer much from the early febrile reactions and the later reactions, which are marked slight, did not affect her at all, the temperature running up towards evening to about 100°, or sometimes a little higher. The wounds left after scraping healed in a few days. On April 2nd the places where the plasters were first applied were allowed to heal, and in the other parts, where the plaster had not been applied, the usual improvement noted in the early part of this treatment was observed. July 6th.—Still under treatment. Improving.
Injections mmenced on ·ch 2nd, 1891. al number of ections up to ril 8th = 45. al amount of id used up to that date ·393 grms.	On March 11th the creosote and salicylic plasters were begun on the right cheek	Note on April 8th.—"The left cheek is still much swollen, red, and scaling, and is improving rapidly as regards the lupus. On the right side the scar is still broken down as the result of the plaster, but is healing at the lower part. The patches on the arm and hand are already much improved. Glands unaltered" *Result.*—Improving	The first five injections were followed by marked general and local reaction, and by a scarlatiniform eruption followed by desquamation. The sore on the leg also reacted, but the old scars in leg and arm did not react. The left cheek healed rapidly, and the right broke down well under the salicylic plasters. Sections of skin taken before the treatment was commenced show an unusual amount of tubercular tissue. Treatment stopped in the beginning of June. Face apparently well. Still remained well in July.

There were thus ten cases of lupus under treatment, of which two have been going on for too short a time to be of value; both are steadily improving. I may say that in all the remaining eight cases improvement occurred at first, and in some it was very considerable; in one indeed (No. 8) there is now hardly any disease to be detected. In four of the eight cases (Nos. 1, 2, 4, and 8) the improvement is maintained or increasing. In one (No. 5) when the patient was last seen the improvement had come to a standstill, although there was still a good deal of lupus tissue present. In three, after the improvement had gone on to a considerable extent, it came to a standstill, and now shows a distinct tendency to relapse, but in none is the disease nearly so bad as before the treatment was commenced. My present view as to the position which this treatment ought to occupy in the treatment of cases of inveterate lupus, is that which it occupies in the treatment of case No. 9. The greatest improvement was obtained in a case (No. 8) treated by the "continuous" plan. Case 1 is very interesting as showing the very remarkable improvement after two severe reactions, but recurrence after leaving off the treatment; this improvement affecting not only the lupus, but also the disease of the metacarpal bone.

DISEASES OF BONES AND JOINTS.

A.—CASES WITH UNBROKEN SKIN.

II.—Diseases of Bones and Joint

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
11	W. P., æt. $3\frac{1}{2}$, female, K. C. H., admitted Dec. 9th, 1890. Discharged Jan. 21st, 1891. *Diagnosis.*—Disease of knee-joint; enlarged glands in neck	No phthisical family history. Four weeks before admission the right knee was noticed to be swollen and painful, and since that time the swelling and pain have rapidly increased. No known cause. No treatment	Internal organs healthy. Enlarged glands on both sides of neck. Right knee much enlarged; measures across the centre of the patella $8\frac{3}{4}$ in.; left measures $7\frac{7}{8}$ in. Breadth of knee below adductor tubercle measured with callipers = on right side $2\frac{5}{8}$ in.; on left side $2\frac{1}{8}$ in. Knee flexed nearly to a right angle, and pain on attempting movement. Synovial membrane much thickened; no fluid in joint. (In this case before the Koch period I would have employed the expectant method)	·002″–·003′; $_1$·005′; $_2$·007 $_1$·008; $_1$·009; $_1$·01; $_4$·01 $_2$·012; $_1$·012; $_1$·015 $_2$·02; $_3$·02; $_2$·02; $_3$·025. Be came out-patient, the dos being rapidly raised to ·] the patient coming up twic a week for injection
12	C. F., æt. 16 months, male, K. C. H., admitted Dec. 11th, 1890. *Diagnosis.*—Disease of knee-joint; enlarged glands in neck	No tubercular family history. Sent for treatment from Guy's Hospital. The disease is said to have begun 4 months ago after a fall	Internal organs healthy. Small enlarged glands on both sides of neck. The left knee is kept flexed, and any attempt to extend it causes pain. Cannot be extended beyond 48°. The joint is much swollen, and there is marked synovial thickening. On the outside of the joint towards the lower part is a fluctuating swelling, which, however, does not seem to communicate with the joint. Circumference of left knee over centre of patella $1\frac{1}{8}$ in. more than the other. By callipers transversely just below the adductor tubercle the left side is $\frac{1}{4}$ in. larger than the right. (When I first saw this case I took a gloomy view of it, and while before the Koch period I would have persevered with expectant treatment for a time, I should have expected that it would come to operation ultimately)	·0015′; $_1$·002′; $_2$·003′; $_1$·00 $_1$·005′; $_1$·006; $_1$·008′; $_4$·01 $_1$·015; $_1$·02′; $_1$·025; $_2$·0 $_3$·04; $_2$·04 (interval of] days); $_1$·01; $_1$·015; $_1$·02 $_1$·03′; $_2$·04; $_1$·05′–·06; $_1$·0 $_1$·07; $_1$·08; $_1$·09′; $_1$·1; $_2$· $_1$·1; $_2$·1; $_1$·1; $_1$·1; $_2$· $_1$·1; $_1$·1; $_2$·1; $_1$·1; $_1$·1; $_2$· $_1$·1–·1; $_1$·1–·1–·1; $_2$·1; $_1$· To be continued

A. *Cases with Unbroken Skin.*

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Injections commenced on Dec. 10th, 1890. Total number of injections up to April 6th, 1891 = 32. Total amount of fluid used up to the same date = 1·438 grms.	Extension till Jan. 13th. Massage afterwards	Note on April 5th.—"Child runs about, and is apparently in good health. Knee normal in size and appearance; no pain. Glands in neck in *statu quo*" *Result.*—Very remarkable improvement. Complete disappearance of thickening and complete restoration of movement. Glands in *statu quo* (In such a case I am afraid I would now recommend the expectant method and not Koch's treatment, for I can see nothing in this case to indicate that it would behave as it did under the Koch treatment and not as the other cases did. While one does occasionally get complete restoration of tubercular joints with expectant treatment, it is very rare where there is such marked thickening as in this instance, and certainly not in the course of 5 weeks)	The patient did not suffer from the injections, and the first reactions were quite slight; in fact, the temperature usually fell to between 96° and 97° a few hours after the injections instead of rising. After the first injection there was a little pain in the knee, but there was none subsequently. The swelling of the knee steadily went down, and when discharged it was only $\frac{1}{5}$th of an inch larger in circumference than the other; the pain also quickly disappeared, and the movements became free. Massage was begun on Jan. 13th, and the patient was allowed to get up on Jan. 20th, when she could stand and walk without pain. She has since been allowed to run about without any local treatment. Treatment stopped in the middle of May. Remains well in July.
Commenced Dec. 12th, 1890. Total number of injections up to April 8th = 46. Total amount of fluid used up to that date = 2·7345 grms.	Extension and sandbags: left off on March 20th, and child allowed to move about	Note on April 5th.—"There is now hardly any perceptible difference between the two knees. The movement is perfect, and the child is standing and kicking in bed all day long. The glands in the neck seem to be in very much the same condition as they were originally" *Result.*—Very remarkable improvement in the condition of the knee. Disappearance of fluid, thickening, and pain; movements perfect. Glands in *statu quo* (I would now treat such a case as I would have done before the Koch period, for as in No. 11 I can as yet find nothing to indicate that it would improve so remarkably as it did under the Koch treatment)	The patient suffered very little after the injections, the chief symptoms being cough. The slight rise of temperature usually occurred on the day after the injections. The interval of 18 days was on account of an acute abscess in the scalp and a secondary one in right parotid region. These were opened and quickly healed. During the first injections the knee increased in size half an inch. On Dec. 27th it was found that the fluctuating swelling on the outer side of the joint had disappeared, and fluid was present in the joint. A drop or two was drawn off for examination, and was found to be of a glairy character with white flakes in it. After this the fluid became absorbed and had disappeared on Jan. 5th, and the synovial thickening steadily disappeared, and the movements of the joint became free and painless. The increase in size transversely was slower in disappearing. Treatment stopped in beginning of May. Have not seen patient since.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount o injections.
13	J. H., æt. 6½, female, K. C. H.; admitted Nov. 26th, 1890. *Diagnosis.*—Hip-joint disease	No tubercular family history. The first symptoms of hip-joint disease were noticed last August, but they became much more marked after a fall 4 weeks ago	Internal organs healthy. Right limb slightly flexed and adducted. Limb can be flexed with care to a right angle, but not beyond. Cannot be quite extended. No rotation or adduction, and attempts at these movements cause great pain. No shortening. Some thickening in front of the joint. Antero-posterior measurements by callipers over the head of the femur give on right side $2\frac{3}{4}$ in., and on left $2\frac{1}{2}$ in. Nothing noticed wrong with the eyes. (Before the Koch period I would have employed the expectant treatment in this case)	·003‴; $_2$·003‴; $_1$·003′ $_3$·002‴; $_1$·002″; $_2$·002 $_1$·002′; $_1$·002′; $_2$·002′; $_4$·003 $_2$·003′; $_1$·004′; $_1$·005–·00 $_1$·008; $_1$·01′; $_2$·01; $_1$·01 $_1$·02: $_2$·025′; $_1$·03′; $_1$·0 $_4$·05′; $_1$·06 (operation, inter of 13 days): $_1$·01; $_2$·02′; $_1$·0 $_1$·04′; $_1$·05; $_1$·06′; $_2$·0 $_2$·08′; $_1$·09; $_1$·1; $_2$·1; $_1$ $_1$·1: $_2$·1; $_1$·1; $_1$·1–·1; $_1$ $_1$·1; $_1$·1–·1–(·1, ·1)–·1–·1– ·1–·1–·1. Treatment co tinued

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Dec. 3rd, 1890. Total number of injections up to April 8th, 1891 = 53. Total amount of fluid used up to the same date = 2·76 grms.	Before the occurrence of the dislocation no retentive apparatus was employed. On Feb. 3rd the ordinary anterior incision was made into the hip-joint. Remarkably little disease was found, the synovial membrane being only very moderately thickened. The ligamentum teres had, however, been completely destroyed, and hence partial dislocation of the head of the femur was readily produced. The cartilages were intact, and there was no evidence of bone disease. As much of the softened synovial membrane as possible was removed, the joint thoroughly washed out, and the head of the bone replaced in the acetabulum. Wound stitched up; no drain. Extension and sandbags. *Partial arthrectomy after 24 injections.* Abscess opened on April 1st (see notes). On microscopical examination of the synovial membrane removed on Feb. 3rd, tubercles were found for the most part much atrophied. Two guinea-pigs were inoculated on Feb. 3rd with portions of synovial membrane and became tubercular	Note on April 5th.—"Patient much more comfortable since abscess was washed out. The dressing has not yet been changed. Anæmic, but otherwise apparently well." *Result.*—Marked improvement at first; dislocation; partial arthectomy; abscess formation. (In such a case I would now employ expectant treatment without using Koch's method. At the same time it cannot be denied that marked improvement followed the use of this method at first, and that the subsequent dislocation, which led to all the trouble, was only due to the fact that the ligamentum teres had suffered from the disease more than any other part of the joint)	As regards the general effect of the injections, the first four caused high temperature, headache, sickness, and coughing. Seven days after the treatment was commenced the child was markedly jaundiced, the stools were white and offensive, and there was bile in the urine. The jaundice passed off in about a week. After the first injection the patient complained of pain in the right eye, and some vesicular keratitis was found. This recurred after the next three injections and then ceased, and the eye remained well. There was a great deal of pain in the hip after the first two injections, and the hip was more swollen, but this did not recur with the later injections. From the end of the third week very remarkable improvement was observed in the hip, the thickening entirely disappeared, and the movements became free and painless. On Jan. 7th the hip-joint seemed normal in all respects, and remained so till Jan. 25th, when the patient was allowed to get up. After being up a short time she complained of severe pain in the joint, and next day it was found that partial dislocation of the head of the femur had occurred; this was reduced under chloroform. On Feb. 3rd the joint was opened as before described. The wound healed by first intention. On April 1st it was noticed that the scar was bulging about the middle. An incision was made into this, and about an ounce of thin pus was evacuated. It did not seem to communicate with the joint. The cavity was scraped out, iodoform and glycerine injected, and the wound stitched. Treatment stopped in beginning of May. Not healed at end of June, but improving under other treatment.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
14	E. S., æt. 5, female, K. C. H., admitted Dec. 1st, 1890. *Diagnosis.*—Disease of hip-joint	Tubercular family history. The disease began last April after a fall. Has been wearing a Thomas's splint for some months	Internal organs healthy. The right limb is somewhat flexed (35°), adducted, and markedly rotated outwards; ¾ in. shortening. There has evidently been a true dislocation of the head of the bone upwards and forwards. A good deal of thickening about the joint, but no sign of abscess. Tenderness on pressure. No movement possible (Before the Koch period I would have excised the head of the bone)	·002″; 2·003‴; 2·003″ 1·003″ 1·004″; 2·005′ 1·005′; 1·006′; 1·007 4·008′; 2·009′; 2·01′; 1·012 2·015′; 3·02″; 2·02′; 6·015 3·015′; 2·02″; 1·02′; 1·0 2·04; 1·05 (operation, inte val of 11 days); 1·01″; 1·0 1·02′; 4·02; 1·03; 2·0 1·05; 1·06; 2·07; 1·0 1·09′–·08–·08–·09–·1–·1; 1· ·1–·1–·1–·1–·1–(·1, ·1)–(·1, ·1 (·1, ·1)–(·1, ·1)–(·1, ·1)– (·1, ·1)–(·1, ·1)–(·1, ·1)– (·1, ·1)–(·1, ·1)–(·1, ·1)– (·1, ·1). To be continued
15	J. F., æt. 8, female, P. G. H.; admitted Dec. 29th, 1890; discharged March 18th, 1891 *Diagnosis.*—Hip-joint disease; enlarged glands	Phthisical family history. Losing flesh since last summer; limping for 2 months; starting at night for 2 weeks. No known cause; no previous treatment	Internal organs healthy. A few enlarged glands in neck and both groins. Pain in right hip and knee when walking. Leg fully extended and slightly adducted. No movement at hip-joint and pain on attempting it. No shortening. Fulness in front of joints. Thickening about trochanter. No thickening felt per rectum. (Before the Koch period I would have performed a partial arthrectomy of the hip in this case with the view of removing the sequestrum, the presence of which I was sure of. That a sinus might have formed after this treatment is not improbable from the results obtained in Nos. 27 and 29)	·002‴; 1·002″; 1·004 1·004; 1·006; 1·008″; ·0 1·01; 2·015; 1·02; 1· 2·035; 1·04; 1·05; ope tion, interval of 14 da 1·015′–·02′–·02–·02′–·025′– ·03′; 1·04′–·04′–·05′–·05′– ·07′; 1·09–·1′; 1·12′; 1·1 1·15–·15′–·2′–·25; 1·3″; Stopped by mistake; sumed on April 7th

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Dec. 4th, 1890. Total number of injections up to April 8th, 1891 = 68. Total amount of fluid used up to that date = 4·252 grms.	On Feb. 10th, 1891, the abscess was opened and thoroughly cleared out and the head of the bone removed. The articular cartilage was partially destroyed. The acetabulum was filled up with soft material which was removed. Wound stitched up; drainage-tube inserted at the upper part of the wound. Extension. Drainage-tube left out 3 days later. *Excision of the hip-joint after 23 injections.* Two guinea pigs were inoculated with material removed, and became tubercular	Note on April 5th, 1891.—"There is a sore where the drainage tube was with tubercular granulations. The middle of the scar is also breaking down. General condition good." *Result.*—Abscess, formation; excision; breaking down of the wound. (In a similar case I would now excise the head of the bone and only employ the Koch treatment if the wound would not heal or if it broke down)	Did not suffer much after the injections, and there was very little local reaction. After the first injections there was a slight papular rash on the face and back, and a good deal of cough. For a time the thickening around the hip diminished, but on Jan. 1st the tenderness returned, and an abscess soon showed itself in front. (This renewal of tenderness was coincident with an injection made directly over the joint instead of into the back.) Excision was performed on Feb. 10th and the wound healed by first intention except where the drainage-tube was. This sore has remained unhealed, and at first extended somewhat, but has not done so lately. On changing the dressing on April 5th it was, however, found that a point in the centre of the scar had broken down. Not healed at end of June, but improving under other treatment.
Injections commenced on Jan. 5th, 1891. Total number of injections up to March 17th = 36. Total amount up to same date = 2·482 grms.	On Feb. 5th the hip-joint was opened by the anterior incision and a large sequestrum was found and removed from the usual seat in the neck of the femur. Some curdy matter in joint, cartilages intact, synovial membrane not much thickened. As much of the tubercular material was removed as possible. Wound stitched up, no drain. Sand-bags on each side of limb. No extension *Partial arthrectomy after 14 injections* Numerous tubercles were present in the synovial membrane. Sent to convalescent home on March 18th	Note on April 7th, 1891.—"Small sinus in middle of scar; leads to bare bone in the neck of the femur; hardly any discharge. Condition of patient good. Glands in *statu quo*" *Result.*—Slight improvement at first; partial arthrectomy; healing of the wound; formation of a sinus after the treatment had been stopped (In such a case I would now perform partial arthrectomy at once and then use the Koch treatment)	After the first injection temperature was highest (104°). After 19 hours there was a good deal of pain in the joint. After the second injection there was no further pain. On 19th Jan. it is noted that there was no pain in the joint, and that slight movement was possible. This mobility increased up to the time of the operation, and the thickening in front of the joint steadily diminished. Operation wound healed by first intention. When sent to the convalescent home on March 18th the injections were stopped by mistake. About the 1st of April a small sinus formed in the middle of the scar. Injections recommenced on April 7th. Treatment stopped in the middle of May. Not healed at end of June.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
16	H. F. C., æt. 5, male, K. C. H., admitted Jan. 20th, 1891. *Diagnosis.*—Disease of hip-joint with abscess	Tubercular family history. Disease began after a fall at the end of 1889. Patient was admitted to hospital in Feb., 1890, and sent out in July wearing a Thomas's splint. In Dec. last a swelling was observed about the trochanter	No disease of internal organs. Right limb slightly adducted; no flexion; no shortening. Slight movements of abduction and adduction and rotation causing pain; no flexion possible. Just above and behind the trochanter is a small abscess. There is a good deal of thickening in front of the joint (Before the Koch period I would in this case have excised the abscess, but not have opened the hip-joint)	$\cdot 002'''$; $_2 001''$; $_1 \cdot 002'''$; $_1 \cdot 003'$; $_2 \cdot 005''$; $_1 \cdot 006$ (operation, interval of 11 days); $_1 \cdot 002$; $_1 \cdot 004$; $_1 \cdot 006$; $_1 \cdot 008$; $_2 \cdot 01$; $_1 \cdot 015$; $_2 \cdot 015'$; $_1 \cdot 02$; $_1 \cdot 03$; $_2 \cdot 04$; $_1 \cdot 05$; $_1 \cdot 06$; $_2 \cdot 07$; $_1 \cdot 08$; $_1 \cdot 09$; $_2 \cdot 1$–$\cdot 1$–$\cdot 1$–($\cdot 1$, $\cdot 1$); $_1 \cdot 1$; $_1 \cdot 1$; $_1 \cdot 1$; $_1 \cdot 1$
17	W. R., æt. 32, male, K. C. H.; admitted Nov. 29th, 1890; discharged Feb. 18th, 1891 *Diagnosis.*—Disease of knee-joint and tibia	No phthisical family history. Disease began about 3 years ago after an injury to his knee (combination of twist and blow). The knee has improved from time to time when he lay up; on resuming work it always became painful again. Lately has been treated by rest and Scott's dressing	Internal organs healthy. Left knee somewhat swollen, more especially at the inner side. There is marked swelling over the inner side of the head of the tibia and tenderness on pressure at that part. The main synovial thickening is just above this. There is a slight amount of flexion without pain; attempts to bend it beyond half a right angle cause pain; the joint cannot be fully extended (Before the Koch period I would have cut down on the inner side of the head of the tibia and removed as much of the affected tissues as possible)	$\cdot 01''$; $_2 \cdot 01'$; $_2 \cdot 015''$; $_1 \cdot 01$[illegible]; $_2 \cdot 02''$; $_1 \cdot 02'$; $_1 \cdot 03'$; $_2 \cdot 04$; $_1 \cdot 045$; $_3 \cdot 04$; $_4 \cdot 05$–$\cdot 06$; $_1 \cdot 08$; $_1 \cdot 1$; $_1 \cdot 12$; $_2 \cdot 15$; $_3 \cdot 15$; $_2 \cdot 2$[illegible]; $_3 \cdot 1$ (operation, interval of 1[illegible] days); $_1 \cdot 05''$; $_1 \cdot 04'$; $_2 \cdot 06$; $_1 \cdot 07'$; $_1 \cdot 1$; $_2 \cdot 1$; $_1 \cdot 12$; $_1$[illegible]; $_2 \cdot 1$. Injections discontinue

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Jan. 25th, 1891. Total number of injections up to April 8th, 1891 = 30. Total amount of fluid used up to the same date = 1·419 grms.	On Feb. 10th the usual anterior incision was made and the hip-joint examined. There was very little synovial thickening, and the cartilages were intact. The abscess was also opened, and did not communicate with the joint. It was thoroughly cleared out. Wound stitched, drainage-tubes being inserted into both wounds On Feb. 15th both tubes were left out *Partial arthrectomy after 6 injections* Tubercles were found in the synovial membrane on microscopical examination	Note on April 5th.—"The wounds are apparently soundly healed and the thickening in front of the joint has disappeared, Patient in good health." *Result.*—Healing of the arthrectomy wound and the abscess. Disappearance of the thickeniug in front of the joint. (I would now treat such a case as I would have done formerly, without employing Koch's treatment)	The first reaction was severe, the chief symptom being rapid breathing; there was no evident local reaction. In this case it was thought advisable to operate at an early stage. The wounds healed rapidly, both being soundly healed by March 11; they remain healed.
Injections commenced Nov. 30th, 1890; discontinued Feb. 18th, 1891. Length of treatment = 80 days. Total number = 28. Total amount of fluid used = 1·945 grms.	On Jan. 23rd an incision was made into the joint on the inner side, the synovial membrane was granular on the surface, but very slightly thickened; a portion was removed for microscopical examination. The incision was continued downwards over the inner side of the head of the tibia, and some cheesy material was found under the periosteum, close to the upper part of the bone. A hole was also gouged in the bone because it was thought that there might be a tubercular deposit in the bone, but only highly vascular inflamed bone was found. The wound was stitched up—no drain. Healing occurred by first intention. Back splint (no splint had been used previously) On microscopical examinations of the tissues removed tubercles, many of them undergoing atrophy were found. In the synovial membranes especially they were very much degenerated	Patient seen on April 1st. and said that the knee was quite comfortable. The waterglass bandage was not removed, but he was given permission to walk with the bandage on. *Result.*—Improvement in all respects for a time Removal of portions of the affected tissue, healing of the wound. (In such a case I would not now trouble to use Koch's method, but would operate in the manner previously indicated)	After the first injection there was a good deal of pain in the knee, some effusion into the joint and enlargement of the part. This soon subsided, and as the treatment went on the enlargement decreased somewhat, the synovial thickening diminshed, and the range of painless movement of the knee markedly increased; in fact, the patient could bend the knee to a right angle without pain. A trace of albumen was found in the urine the day after the first injection, but disappeared next day and did not recur. Patient was allowed to walk about on Jan. 12th, but this was followed by recurrence of swelling, slight pain, and fluid in the joint. These symptons subsided on rest, but as the tibia now remained tender, the operation was performed. The patient was sent home on Feb. 18th, the whole limb being put up in waterglass. He returned on March 16th and the waterglass was removed. The line of incision remained soundly healed, and was somewhat drawn in at its centre. Knee still somewhat larger than the other, but quite free from pain or tenderness. There was an obscure sense of fluctuation at the inner side of the knee in the neighbourhood of the incision. Waterglass reapplied.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
18	P. C., æt. 5, male, K. C. H.; admitted Nov. 30th, 1890; discharged Feb. 20th, 1891; re-admitted on March 23rd, 1891 *Diagnosis.*—Disease of knee-joint; enlarged glands in neck	No tubercular family history. The disease began at least 6 or 8 months ago. Has not been treated	Internal organs healthy. An enlarged gland on right side of neck. The knee is flexed, swollen, and very painful on any attempt at movement or pressure. The synovial membrane is moderately thickened and there is apparently enlargement of the internal condyle of the femur. The knee is $\frac{3}{4}$-inch larger in circumference than the other (Before the Koch period I would have persevered in the expectant treatment in this case)	·002″; $_1$·003′; ·003′; $_1$·004′: $_2$·005; $_1$·006; $_2$·007; $_1$·009; $_4$·01–·012; $_1$·015; $_1$·02; $_1$·03; $_2$·04; $_1$·04; $_1$·05; $_2$·06; $_3$·07; $_2$·07; $_1$·07; $_1$·08; $_2$·08; $_1$·08; $_1$·1; $_2$·1; $_1$·1; $_1$·1; $_3$·1; $_1$·1; $_1$·1; $_1$·1; $_2$·1; $_1$·1. Sent to convalescent home on Feb. 20th, and while there had 7 injections of ·1. On his return on March 23rd the treatment was discontinued
19	W. J., æt. 9, male, K. C. H., admitted Dec. 12th, 1890 *Diagnosis.*—Disease of knee-joint	No tubercular family history. Five weeks before admission fell on to his right foot off a tree, and felt pain in the right knee, which began to swell at once, and has steadily increased in size and become more painful. Has been treated by rest and splints	Systolic murmur at apex and base of heart; dilatation. Other organs healthy. On admission the right knee-joint was markedly swollen, being $1\frac{1}{8}$ in. larger in circumference than the left over the centre of the patella. No fluid in joint. Tender on pressure. Joint cannot be completely extended, but can be flexed rather beyond a right angle, but flexion is painful. Patient was kept at rest in bed for 19 days, and during that time the swelling of the knee increased $\frac{1}{8}$th of an inch (Before the Koch period I would have given a further trial in this instance to absolute immobilisation, &c., but I have no doubt that before many weeks had elapsed complete arthrectomy would have been necessary)	·002″; $_3$·002‴; $_2$·002‴; $_2$·002″; $_2$·002′; $_1$·003″; $_1$·003′; $_1$·004′; $_1$·005″; $_1$·005′; $_2$·007′; $_1$·008″; $_1$·008′ (operation, interval of 16 days); $_1$·002″; $_1$·003′; $_1$·005′; $_2$·005′; $_1$·008′; $_1$·009″; $_2$·01′; $_1$·015″; $_1$·02″; $_1$·02″; $_1$·02; $_1$·03′; $_2$·04′; $_1$·05″; $_2$·05′; $_1$·06′; $_1$·06; $_1$·06–(·06, ·06)–·06–·07′; $_1$·80; $_1$·09′; $_2$·1. To continue

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Dec. 8th, 1890; discontinued on March 23rd, 1891. Total number of injections =39. Total amount of fluid used =2·366 grms.	Extension and sand bags. Sent to convalescent home on Feb. 20th, 1891, with the leg in plaster of Paris, and to go on with injections. On March 31st complete arthrectomy was performed. The synovial membrane was much thickened, and the articular cartilage was eroded, especially over the lower part of the inner condyle of the femur and the inner tuberosity of the tibia. Several cheesy foci were found in the tibia at this part. Wound stitched up; no drain. *Complete arthrectomy after 39 injections.*	Note on April 8th, 1891.—"Patient is very comfortable. The dressings have not yet been changed. Glands in neck quite small." *Result.*—Improvement for a time as regards pain, and to some extent as regards swelling. Abscess formation: complete arthrectomy. Glands smaller. (In such a case I would now persevere with expectant treatment, and not employ Koch's method)	Nothing noteworthy as regards the reactions, with the exception of a papular rash in patches on the back and sides after the first injection. The knee swelled and became more painful after the first injection, but this soon subsided, and on Jan. 13th it is noted that there was no pain on jarring or moving joint, and the knee was only ¼ inch larger in circumference than the other. The improvement as regards the thickening then seemed to come to a standstill. On Feb. 20th the limb was put up in plaster of Paris, and he was sent to a convalescent home to have ·1 injected twice a week. At that time movement of the joint was painless, and it was ¼ inch larger than the other, the chief thickening being about the internal condyle. When the patient was readmitted on March 23rd, an abscess was found on the inner side of the knee, and there was considerable pain on movement. On March 31st complete arthrectomy was performed
Commenced Dec. 31st, 1890. Total number of injections up to April 8th = 38. Total amount of fluid used up to that date = 1·04 grms.	On Feb. 3rd the joint was opened by long vertical incisions on each side of the patella; the synovial membrane was markedly thickened, but there was not nearly so much soft material as in the other cases; portions were clipped away, and as much removed from the surface by washing out, rubbing with sponges, &c., as possible, but much tubercular material was left behind. Stitched: no drain; back splint. *Partial arthrectomy* after 13 injections. The wounds healed by first intention. Two guinea-pigs were inoculated with portions of synovial membrane on Feb. 3rd, but have not become tubercular; numerous tubercles were present in the synovial membrane	Note on April 6th.—"The wounds appear to be soundly healed, but there is still much synovial thickening. No pain, and patient can lift leg off the bed without pain." *Result.*—Slight improvement; partial arthrectomy; healing of wound; disappearance of pain. Thickening of synovial membrane still remaining, but not increasing. (At the present time I would treat such a case as before indicated, reserving the Koch treatment in case the wounds would not heal. At the same time there is little doubt that advantage has been gained by the treatment, for without it I feel sure the wounds would have broken down after the partial arthrectomy. Whether the treatment will have been ultimately of advantage in this case cannot yet be decided)	The first 2 injections caused pretty severe symptoms, chiefly cough, sickness, and headache. There was also a good deal of pain in the knee after the first 2 injections, and after the first it increased 2 inches in circumference. It, however, very quickly went down to and below its original size, and on Jan. 14th it was ¾ inch less than it was originally. It, however, increased again, and before the operation on February 3rd the measurement was the same as the original one. The wounds healed by first intention. Complete arthrectomy performed in the middle of May. Healed.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
20	F. G., æt. 3, female, K. C. H.; admitted Dec. 2nd, 1890 *Diagnosis.*—Disease of elbow-joint	No tubercular family history. Disease began 18 months ago after an injury. Has been treated at various hospitals, but elbow has lately got worse, in spite of rest	Internal organs healthy. Right elbow-joint 1½ inches larger than the other, measured over bend of elbow; the chief swelling is on outer side of arm, especially over the head of the radius. The movements of flexion and extension are limited and pronation and supination are impossible. There is pain on movement (Before the Koch period I would have persevered with expectant treatment for some time, but I believe that complete arthrectomy would ultimately have been necessary)	·002‴; ₂·002‴; ₃·002‴ ₁·002‴; ₁·002″; ₂·002′ ₁·003′; ₁·004′; ₁·003; ₄·004′ ₂·005′; ₁·007; ₁0·1′; ₂·012″ ₁·015′; ₁·02′; ₂·015′; ₁·02′ ₁·02 (operation, interval o 11 days); ₁·02″; ₁·02′ ₂·03′; ₁·03′; ₁·04′; ₂·05′ ₁·06′; ₂·07′; ₁·07; ₁·08 ₁·09; ₂·1; ₁·1; ₂·1; ₁·1 ₁·1; ₁·1–·1–·1–·1–·1–·1; ₁·1 ·1–·1–·1–·1–·1–(·1, ·1)–(·1, ·1) (·1, ·1)–(·1, ·1)–(·1, ·1)–(·1, ·1) (·1, ·1)–(·1, ·1)–(·1, ·1)–(·1, ·1) (·1, ·1)–(·1, ·1)–(·1, ·1)–(·1, ·1) (·1 ·1)–(·1, ·1)–(·1, 1). To b continued
21	M. G., æt. 6, female, K. C. H., admitted Dec. 3rd, 1890. *Diagnosis.*—Disease of elbow-joint with abscess	Tubercular family history. The disease of the left elbow-joint has been noticed for the last 3 months, attributed to a fall. No treatment	Internal organs healthy. There is general thickening of the synovial membrane of the left elbow-joint, but not very marked. Great rigidity of the joint; hand completely pronated. Pain on attempting movement, also tenderness on pressure on the elbow. There is a small abscess the size of a hazel-nut over the back part of the head of the radius; no fluid in the joint. (Before the Koch period I would have performed complete arthrectomy at once in this case)	·003‴; ₂·003‴; ₂·003″ ₂·003‴; ₁·003′; ₂·004′ ₁·004′; ₁·005; ₂·007′; ₃·008 ₂·009′; ₁·01; ₁·012′; ₃·015 ₃·02″ (abscess excised, in terval of 9 days); ₁·025′ ₁·025″; ₁·03″; ₂·04″; ₁·04′ ₄·04′; ₁·04′; ₁·06′; ₂·07′ ₁·08′; ₁·1′; ₂·1″; ₁·1; ₁·1 ₁·1; ₂·1; ₄·1; ₁·1; ₁· Treatment stopped

Total number, mount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
ommenced on ec. 3rd, 1890. otal number of injections up to April 8th, 1891 = 81. otal amount of uid used up to at date = 5·81 grms.	Till the operation no retentive apparatus was applied, On Jan. 23rd incisions were made on the inner and outer sides of the joint, and a good deal of soft tissue was removed, but I intentionally did not clear the	Note on April 5th, 1891.—"There is a granulating patch about the size of a shilling in the middle of each scar, but the granulations are much healthier than they were, and considerable healing has occurred during the past week. General condition of the patient good" *Result.*—Improvement for a time. Partial arthrectomy. Healing and subsequent healing down of scars. Lately healing occurring (In a similar case I would now persevere with expectant treatment for a time without using Koch's method)	The first reactions were somewhat severe, the chief trouble being cough and sickness, and after the first injection the arm became painful and more swollen, but this did not occur subsequently. The elbow became more moveable and painless, and the swelling diminished somewhat (to about ¾ in. more than the other). As, however, matters became stationary, the joint was opened as before described. The wounds healed up, but on Feb. 15th it was found that the scars were beginning to break down in two places, and this continued till about half of each of the scars had broken down. Since that time the condition for a time remained stationary, but quite recently healing has been going on.
int out so thoroughly as I would herwise have done. The wounds ere stitched up; no drain. Ex- rnal splint with shoulder-cap. *rtial arthrectomy after* 19 *injec- ons.* bercles found in synovial mem- ane on microscopical examina- on. Two guinea-pigs were in- ulated with portions of synovial embrane on Jan. 23rd, and be- me tubercular			
Commenced ec. 4th, 1890. opped March 8th, 1891. ntinued for 94 days. tal number of jections=34. tal amount of id=1·439 grms.	No splint was employed in the first instance. On Jan. 16th the abscess over the head of the radius was cut out and found to consist of a mass of cheesy material with very little fluid in it. Wound left open. On March 10th	Note on April 6th.—"The wounds have healed by first intention except on the outer side where the drainage tube was. This was left out to-day. Patient in good health" *Result.*—No improvement. Complete arthrectomy. Healing of wound (In a similar case I would now perform complete arthrectomy at once, and only use the tuberculine if necessary. I can see no advantage from the treatment in this case)	The constitutional effects of the injections were chiefly the fever, headache, and a little tendency to sickness. After the first injection there was a good deal of pain and swelling in the elbow joint, but this did not recur. It seemed as if a little hemorrhage had occurred into the abscess as the skin over it became black and blue. For a time the movements of the elbow became painless, and slightly increased in range, and the measurement of the elbow became slightly less, but the improvement was very little marked and soon came to a standstill. On Jan. 16th the abscess was removed. The wound healed very slowly but had healed by March 8th. In the beginning of March it was found that the elbow had greatly increased in size and that a quantity of fluid was present in the joint, and on March 10th complete arthrectomy was performed.
mplete arthrectomy was per- med. A quantity of cheesy pus as found in the joint, the syno- al membrane was much thickened d broken down, the articular rtilages extensively destroyed, d a soft caseating deposit in the ver part of the greater sigmoid vity, which had evidently been e starting-point of the disease. ound stitched up; drain inserted the outer side 3 days later. *plete arthrectomy after* 34 *in- tions.* guinea-pigs were inoculated on a. 16th with the wall and con- nts of the abscess and became bercular			

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
22	F. C., æt. 1½, female, P. G. C. H., admitted Dec. 5th, 1891. *Diagnosis.*—Disease of elbow-joint; subcutaneous tubercular nodule; enlarged glands in neck; corneal ulcer	Phthisical family history. Disease of right elbow began about a month before admission, attributed to fall. No previous treatment. Subcutaneous nodule over outer side of left patella noticed for 14 days. Corneal ulcer on right eye noticed for 7 days	Rickety child. Internal organs healthy. Enlarged glands in neck. Right elbow much swollen, the swelling involving the whole region of the joint. Fluctuation on inner and outer sides. Measurement over olecranon 1¼ inches more than on other side. Arm somewhat extended (angle of 100°). Movement through angle of 15°. Small subcutaneous nodule over outer side of left patella, size of a bean; skin over it red and adherent. Peripheral corneal ulcer on right eye. (In such a case before the Koch period I would have performed complete arthrectomy at once, and have removed the nodule over the knee)	·0015‴; $_1$·0015‴; $_1$·0015‴; $_1$·0015″; $_2$·002″; $_1$·003′ $_3$·003′; $_2$·004′; $_1$·005′ $_1$·005′; $_2$·007″; $_1$·008′ $_2$·009″; $_1$·01″; $_1$·01′; $_2$·015 $_1$·012″; $_1$·012 (operation, i terval of 12 days); $_1$·01′ $_1$·012″; $_2$·012″; $_1$·012′ $_2$·015″; $_1$·02″; $_1$·02″; $_1$·02 ·02–·02′–·02′–·02–·02–·03; $_1$·03″–·03–·03–·04″–·04″; $_1$·04″; $_1$·04″–·05′–·06′–·07 ·08″; $_2$·1″–·1′–·1′–·15′–·2′–·2 $_1$·25‴; $_2$·2′; $_1$2′; $_1$·25 $_1$·2″–·2. Treatment disco tinued
23	C. K., æt. 3, male, K. C. H., admitted Dec. 1st, 1890; discharged March 18th, 1891 *Diagnosis.*—Strumous dactylitis; enlarged cervical glands; sinus in buttock	Tubercular family history. A year ago patient had an abscess in the left buttock and a sinus has remained ever since. Strumous dactylitis first noticed last August	Internal organs healthy. At the lower part of the left buttock is a septic sinus into which a probe passes for about 2 inches but does not go to bone. There is a typical strumous dactylitis of the left ring finger, affecting the first phalanx. No sign of suppuration but great tenderness on pressure (Before the Koch period I would have cut out the sinus in the buttock; have put the finger at rest and employed the usual remedies)	·002‴; $_2$·002‴; $_1$·002 $_1$·003″; $_3$·003″; $_2$·003; $_1$·00 $_1$·005″; $_1$·005; $_4$·006; $_2$·00 $_1$·007′; $_1$·007–·008; $_1$·0 $_3$·012; $_2$·012; $_5$·015′; $_1$·02 $_1$·02′; $_2$·03′; $_1$·03; $_1$·04′; $_2$·0 $_1$·06; $_1$·08′; $_2$·08′; $_1$·0 $_1$·1′; $_2$·1″; $_1$·1; $_1$·1′; $_2$· $_1$·1; $_1$·1. Treatment stopp

l number, nt, &c., of jections.	Additional treatment.	Condition when last seen.	Notes.
mmenced ec. 9th. number of tions = 53. amount of id used ·799 grms. tment dis- tinued on 24th, 1891	On January 22nd the elbow was opened on the inner and outer sides. No fluid was present, but a mass of caseous material and softened synovial membrane was found and scraped out. The removal of the tubercular	Note on April 6th, 1891.—"The eye is quite well. The sore over the elbow is much smaller and healthier than it was, but still leads into the joint. Glands *in statu quo*. Slight purulent discharge from both ears" *Result.*—No improvement; scars broke down after the arthrectomy	After the first injection the temperature reached 104.6°, and the child was pretty ill; there was local reaction in the elbow and the nodule; nothing special about the eye. The condition of the eye rapidly improved and the ulcer had healed by Dec. 23rd. The nodule over the knee diminished to the size of a pea and then remained stationary. After the operation the incision on the inner side of the elbow healed by first intention, and remains healed. That on the outer side healed by first intention, but began to break down on Feb. 16th. Ultimately the whole scar broke down, but began to heal when the higher doses were reached. On March 16th about 30 seconds after the injection of ·25 grms. the child began to scream, became very flushed all over the body, and immediately afterwards became very markedly cyanosed; the pulse became imperceptible. This passed off within 5 minutes and the child seemed well. The temperature quickly rose, and was 104·4° two hours after the injection. Two hours subsequently it fell to 101°, but subsequently rose and reached 104° in the evening. It came down to normal next evening. On the evening of the injection there was a scarlatiniform rash all over body and legs, which faded next day and was not followed by desquamation. The treatment was discontinued on March 24th, because a small abscess formed in the back and the temperature became irregular. On Dec. 18th there was a slight sero-purulent discharge from both ears, which still continues. A small abscess formed at one side of the rectum, burnt on Jan. 19th, and healed in 10 days.
e was not thoroughly carried Wounds stitched up. No . The nodule over the knee also cut out and was found to thin-walled cyst (tubercular r microscope) containing a of pus *l arthrectomy after* 18 *injec-*		(In a similar case I would at the present time perform complete arthrectomy at once, and leave the question of Koch's treatment till I saw whether the wounds healed or not. In this particular case a better result would in all probability have been obtained in that way)	
rous tubercles were found in ynovial membrane on micro- cal examination. Two guinea were inoculated on Jan. 22nd, became tubercular		April 20th.—Quite lately the temperature has become continuously high, and consolidation of the lungs is occurring (? tubercular). Died in May. No P.M. allowed	
mmenced 3rd, 1890. ed March 1st. inued for days. number of ons = 35. amount of d used ·13 grms.	On Jan. 16th an abscess on the finger was opened and a little cheesy stuff scraped out of bone. Wound left open. This did not heal. On March 3rd the sinus in the finger was laid open and a quantity of cheesy material was removed, and with it portions of bone, including the proximal end of the first phalanx. Wound left open Two guinea pigs were inoculated with the material removed on Jan. 16th and became tubercular	Note on April 6th, 1891.—"Condition of finger remains about the same; an opening still persists and there is a good deal of swelling around it. The sinus in the buttock is again almost entirely healed, and the glands are decreasing in size. General condition good" *Result.*—Temporary improvement as regards finger; subsequent abscess formation. Healing of sinus and diminution in size of glands; temporary relapse as regards these 2 points after stopping the treatment (At the present time I would treat such a case as I would have done before and not employ the Koch treatment. The sinus could have been got more quickly and certainly well by dissecting it out, and the treatment was of no apparent advantage as regards the finger)	After the first 2 injections the temperature rose to 105°, but the child was not markedly ill. The sinus in the buttock discharged freely and the skin around became red and swollen. The finger also became enlarged and more painful. On Dec. 15th the sinus in the buttock was found healed and has remained well since. For a time the finger improved, it became painless and the swelling diminished somewhat, but at the beginning of Jan. signs of abscess formation appeared, and an incision was made on Jan. 16th. This did not heal but presented the ordinary appearance of a tubercular sinus, and was therefore again operated on on March 3rd, and the injections were discontinued. At that time the sinus in the buttock was healed and the glands in the neck smaller. About the middle of March the sinus in the buttock broke down and the glands on the right side of the neck and above the right elbow began to enlarge.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount injections.
24	P. A. S., æt. 5¼, male, P. G. C. H., admitted Dec. 9th, 1890; discharged March 5th, 1891 *Diagnosis.*—Strumous dactylitis; phthisis	Tubercular family history. Disease began 3 years ago. Patient has been operated on twice, the interior of the bones being thoroughly scooped out on each occasion. These wounds have healed. Glands in axilla have also been removed.	Signs of consolidation with crepitation at left apex. Other organs healthy. Proximal phalanges of middle and ring fingers of left hand much enlarged. Small unopened abscess on outer side of middle finger. Fingers tender on pressure	·002‴; $_2$·002″; $_1$·00 $_1$·002″; $_2$·002″; $_1$·0 $_1$·004″; $_1$·004″; $_1$·0 $_2$·005; $_1$·007; $_2$·01′; $_1$·0 $_2$·015; $_1$·02; $_1$·025′; $_2$ $_1$·035; $_1$·04; $_2$·05; $_1$ $_1$·06; $_2$·06′; $_1$·06′; $_1$ $_2$·07′; $_1$·07; $_1$·07′; $_2$ $_1$·07 (operation, interval days); $_1$·07; $_2$·08; $_6$·1″;
25	C. B., æt. 38, female, K. C. H., admitted Feb. 5th, 1891. Discharged April 11th, 1891 *Diagnosis.*—Spinal disease	Marked tubercular family history. When 7 years old suffered from right hip-joint disease commencing after a fall, and she did not recover completely from this till she was 20 years of age. (Suppuration did not occur, and no operation was performed.) Since that time she has remained well till 6 months ago, when she fell and hurt her back, and has suffered pain there ever since, but has had no efficient treatment for it	Internal organs healthy. The right leg is adducted (20°), flexed (55°) and 1 inch shorter than the left, the shortening being entirely limited to the femur. The movements at the hip are extremely limited. There are well-marked signs of spinal disease and considerable antero-posterior curvature of the spine at the lower dorsal region, commencing at the 6th dorsal, ending at the 10th dorsal vertebræ, and most marked opposite the 9th dorsal. Great tenderness on pressure on spines and transverse processes. Pain on stooping, and aching when standing or sitting. No sign of abscess	·002–·003; $_1$·005′; $_1$·0 $_2$·01″; $_1$·01′; $_2$·01′–·01– ·015; $_1$·015; $_2$(·02, ·02)– (·02, ·02)–(·02, ·02)–(·02, · (·02, ·02)–(·03, ·03)–(·03, · (·04, ·04)–(·04, ·04)–(·05, · ·06–(·06, ·06)–06–(·07, · (·08, ·08)–(·09, ·09′)–09– ·1–(·1, ·1)–(·1, .1)–(·1, ·1) (·1, ·1)–(·1, ·1)–(·1, ·1)– (·1, ·1)–(·1, ·1)–(·1, ·1)– (·1, ·1)–(·1, ·1)–(·1, ·1)– (·1, ·1)–(·1, ·1)–(·1, ·1)– (·1, ·1). To continue

tal number, ount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
ommenced c. 9th, 1890. Treatment ped on March 9th, 1891. al number of ctions = 34. al amount of fluid used 1·283 grms.	On Feb. 16th the small abscess on middle finger scraped out; contained only a little cheesy material. Wound left open. Healed readily	Note on April 6th.—"Fingers in much the same condition as before the treatment except that there is now no abscess. General health good; lungs normal" *Result.*—Fingers very much the same. Phthisis much improved	Nothing special to note about the reactions; there was marked swelling of the fingers at first, but this soon subsided and they became less swollen and painless. The abscess, however, persisted, and was therefore scraped out on Feb. 16th. Since then the condition of the fingers has remained stationary.
ommenced . 10th, 1891. of injections o April 10th, 1891 = 76. al amount of d used up to the same ate = 4·888 grms.	Absolute rest on the back, fixed by means of sandbags. Sayre's jacket applied on April 4th	On April 3rd, 1891, there was entire absence of pain or tenderness about the back, and the patient expressed herself as feeling quite well. She was put up in a Sayre's jacket preparatory to her discharge from the hospital *Result.*—Complete loss of pain or tenderness in the spine (How far the same result would have been got without the Koch treatment it is of course impossible to say. I think, however, that it is hardly worth while employing the Koch treatment in these cases unless it is found that absolute rest, &c., fail to arrest the progress of the disease)	The constitutional effects of the injections were slight, but she complained of pain in the spine after each of the early injections. She also had a good deal of diarrhœa after the 4th and 2 subsequent injections, but this passed off completely. She also at this time complained of a little pain in her hip, but this did not recur. The pain in the spine recurred from time to time as the injections were pushed, but there was no general reaction, and for the last few weeks there has been no pain or tenderness. Back still well when seen in June. The treatment was given up when she left hospital.

We had twelve cases of disease of the larger joints with unbroken skin which were treated by Koch's method in the first instance, and of these two (Nos. 11 and 12) have apparently completely recovered; how far this recovery will be permanent time alone will show. The remaining ten required operation; of these seven would have been operated on, at or soon after admission, before the Koch period, leaving three in which the conditions, which determined operation, appeared during treatment. In one of these three cases (No. 13) the determining cause of the operation was the occurrence of dislocation, but I cannot say that operation was absolutely essential in this instance; in another (No. 20) the determining cause was cessation of improvement, and here also the operation at that time was probably not essential, though I have no doubt that it would have ultimately been required (I operated in these cases under the impression conveyed by Koch's paper that the tubercular material would be found broken down and easily removeable, and that in this way operation would expedite the cure); in the third case (No. 18) operation was rendered necessary by the occurrence of suppuration.

Of the operations performed excision was done in one case (No. 14); the scar has here partially broken down. Complete arthrectomy was done in two instances (Nos. 18 and 21), and it is as yet too early to say whether or not there will be recurrence. Partial arthrectomy (*i. e.* partial removal of the affected tissue) was performed in seven cases, and of these three remain healed, and recurrence has taken place in four; of these four cases two are now improving again (Nos. 20 and 22) as regards the local condition, and the other two have broken down quite recently (Nos. 13 and 15); in Nos. 20 and 22 the rapid recurrence was no doubt to some extent my fault, because I did not take pains to remove all the loose tubercular material from the joint, being then under the influence of the view that the tuberculine caused caseation of the tubercular tissue, and that, therefore, it would be readily removed by scraping and irrigation. It is worthy of note that in the

case in which the partial arthrectomy has given the most promising result (No. 16) the operation was performed after six injections had been given, that is to say, the Koch treatment followed rather than preceded the operative interference.

In one case of acute spinal disease all symptoms disappeared in a few weeks, but here absolute rest was also employed.

In the two cases of dactylitis I cannot say that any advantage was derived from the treatment as regards the fingers. In one case, however (No. 23), a sinus which had remained open for a year healed almost at once, and the enlarged glands in the neck diminished in size; and it is very interesting to note that soon after the treatment was stopped recurrence took place in these parts (this recurrence being, however, apparently only temporary), as if the use of the fluid had been keeping the disease in check.

B. *Cases with Aseptic Sinuses, or v*

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount injections.
26	E. C., æt. 15, male, K. C. H., admitted Oct. 2nd, 1890. Discharged Jan. 27th, 1891 *Diagnosis.*—Tubercular disease of os calcis and of the sheath of one of the extensor tendons	No tubercular family history. The disease in the ankle began at the end of 1888, after a kick, and an abscess formed on the outer side of his foot and burst. At the end of 1889, he was again kicked, and a fresh abscess formed in the same situation. In 1888 he also had abscesses at the back of the left leg. About 15 months before admission he had a blow on the knuckles of the right hand followed by abscess, which has never healed. Patient was first admitted on Jan. 30th, 1890; the sinuses on the outer side of the os calcis were opened up and a quantity of cheesy material scooped out of the bone. The ulcer on the hand was also scraped. As the latter did not heal it was scraped again on March 21st, and a portion of the thickened sheath of the extensor tendon of the 3rd finger was cut away. The latter wound healed, but the sore at the knuckle did not. The patient was readmitted on Oct. 2nd, 1890, and on Oct. 4th the os calcis was again scraped and gouged out, and a hole was made through and through the bone with an opening below the internal malleolus; the hand was also scraped	Internal organs healthy; 2 sinuses, one on the inner and one on the outer side of the right os calcis. A probe can be passed right through the bone, and a good deal of bare bone can be felt. There is also a small ulcer over the knuckle of the 3rd finger of the right hand, but the scar along the tendon seems quite sound	·01″; $_2$·01″; $_1$·01″; $_1$· $_1$01″; $_1$·01′–·015″; $_1$· $_1$·015′; $_1$·015; $_2$·02; $_1$· $_1$·03; $_1$·04; $_4$·05–·06; $_1$ $_1$·1; $_4$·12; $_3$·15; $_2$·1; $_2$·12. Treatment dis tinued
27	C. N., æt. 5, male, P. G. C. H.; admitted Dec. 4th, 1890 *Diagnosis.*—Hip-joint disease with sinus; previous partial arthrectomy	Phthisical family history. Disease of right hip began Oct. 1889; no known cause. Was admitted first in Feb., 1890, and arthrectomy was performed on Feb. 20th. A sequestrum was removed from the neck of the femur. The head was partially dislocated, and was replaced. Acetabulum filled with soft material. Cheesy material in joint, and the synovial membrane had cheesy points in it. As much of the diseased tissues was removed as possible. The wound never healed completely, and part of the scar subsequently broke down. Very little progress had been made up to the date of admission, although he had been in a convalescent home for several months	Internal organs healthy, Hip stiff in good position. Sinus in scar leading to joint with flabby granulations. Very little discharge. Much thickening around joint (Before the Koch period I was several times on the point of excising the child's hip joint, and I have no doubt I should ultimately have done so)	·003‴; $_2$·003″; $_1$·00 $_1$·002‴; $_2$·003″; $_1$ $_1$·005″; $_1$·005″; $_2$·0 $_1$·007″; $_2$·008; $_2$·01″; $_1$· $_2$·015; $_1$·02′; $_2$·025″; $_1$· $_1$·02; $_2$·02; $_2$·03: $_2$ $_1$·05′. Injections of ·06 tinued twice a week

Sinuses which have been rendered Aseptic.

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Nov. 26th, 1890; Discontinued n Jan. 21st, 1891. Length of treatment = 56 days. Total number of injections = 23. Total amount of fluid used = 1·135 grms.	None.	Patient was seen on March 25th, 1891. The sinus in the foot was in the same condition as when he was discharged. There was some increased thickening over the knuckle and a small crust, which has formed within the last few days *Result.*—Sinus in foot remained unhealed, but the bare bone disappeared: apparent disappearance of the tubercular tenosynovitis. Relapse of the latter after cessation of treatment (In this case I do not think that any real advantage was gained by the Koch treatment)	Nothing noteworthy as to the general reactions. There was never any marked reaction about the foot; there was at first a little redness and an increased discharge. The sore over the knuckle, however, reacted markedly, the partially healed cicatrix breaking down, but there was no effect where the tendon sheath had been removed. On Dec. 27th no bare bone could be felt on probing the sinuses in the foot, and the sore over the knuckle had completely healed.
Commenced)ec. 9th, 1890.)tal number of jections up to pril 8th, 1891 = 42.)tal amount of uid used up to he same date = 1·515 grms.	Sent to convalescent home on Feb 27th, 1891	Note on April 7th, 1891.—"Sinus apparently soundly healed. No thickening. Child in good health" *Result.*—Great improvement. Rapid healing of the sinus, and disappearance of the thickening. (This is the sort of case in which I think that Koch's treatment will be of great value, and I would adopt it again under similar circumstances)	During the first reaction the temperature reached 104°4 ten hours after the injection, and next day about the same time it again reached 104°: there was a good deal of local reaction. The most severe reaction was after the third injection, but there were no dangerous symptoms. On the 13th of Jan. it is noted that the sinus is soundly healed, and all thickening has disappeared. Since that time the scar has steadily contracted. At the middle of March was allowed to get about in his Thomas's splint. Treatment stopped in April. Remained well in July.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and causes o injections.
28	S. W., æt. 13, female, K. C. H.; admitted Oct. 6th, 1890; discharged Feb. 20th, 1891, to be treated as out-patient *Diagnosis.*—Spinal disease with aseptic sinuses in the right lumbar and iliac regions	No tubercular family history. About 5 months before admission patient began to feel aching pain in the back and was always wanting to lie down. About a month ago she noticed a swelling in her right side. No known cause. When admitted there was slight curvature involving the 12th dorsal, and the 1st, 2nd, and 3rd lumbar vertebræ, and there was pain on pressure over these spines and transverse processes. There was a large lumbar abscess on the right side, and also a large fluctuating swelling in the right iliac fossa reaching as far as the middle line and as high as the umbilicus. The two abscesses communicated. On Oct. 14th both abscesses were opened, thoroughly scraped and washed out, 2 oz. of a 10 per cent. emulsion of iodoform in glycerine injected, and the wounds stitched up. Both wounds afterwards broke down and sinuses remained. On Nov. 4th drainage-tubes were inserted, and a second opening made behind corresponding to the hole in the fascia through which the pus had made its way. The drainage-tubes were removed at the beginning of December	Internal organs healthy. There are 2 openings in the right lumbar region and one just above Poupart's ligament. A long bullet probe goes in for its full length into all the sinuses. There is not much discharge, but the wounds show no sign of healing. Curvature and pain on pressure over the spine as before mentioned (In this case before the Koch period I would have adopted one of two courses, either to continue to treat the sinuses aseptically, the average duration of the case till healing is complete being on an average 8 months, or to scrape out the sinuses again, wash them out, inject iodoform and glycerine, and stitch them up, hoping in this way to obtain union by first intention. The latter is what I intended to do)	·005″; $_1$·005″; $_2$·006 $_1$·005″; $_1$·005″; $_4$·006 $_1$·007; $_1$·008′; $_2$·01; $_1$·0 $_1$·012; $_2$·012; $_1$·015; $_1$·0 $_2$·03; $_1$·04′; $_1$·06; $_2$·0 $_1$·12; $_1$·12′; $_2$·12; $_1$·1 $_1$·1; $_2$·1; $_1$·1; $_1$·1; $_1$·12- Sent to convalescent ho for a month, where ·1 w injected twice a week. Sin her return on March 2 ·1 has been injected thr times a week
29	T. K., æt. 5½, male, P. G. C. H., admitted Feb. 10th, 1891 *Diagnosis.*—Hip-joint disease with sinus; previous partial arthrectomy	No phthisical family history. Right hip-joint disease for 14 months; no definite cause. Was admitted first in Feb. 1890, and arthrectomy was performed on Feb. 7th. A sequestrum was removed from the neck of the femur, and as much as possible of the capsule was taken away. The cartilages were intact. Wound healed by first intention except at middle, where a small sinus remained, which healed at the end of April. This remained healed till Sept. 3rd, when a probe could be passed in for a considerable distance	Internal organs healthy. Hip-joint stiff, slightly adducted and everted, completely extended. No shortening. Some thickening felt in front of joint. Sinus leads down to bare bone in neck of femur; very little discharge. A good deal of thickening in front of the joint and enlarged glands in both groins. A few small enlarged glands in neck (Before the Koch period I would in this case have persevered with antiseptic dressings for some months, and had the sinus not closed I might then have operated)	·002‴; $_3$·002‴; $_1$·002 $_1$·002″–·002–·002–(·002, ·0 $_1$·002–(·003, 003′)–·005– (·005′, ·005)–·008–012; $_1$·012–·013–·02–·035′–·05′– ·07–·1″–·1′–(·1, ·1′)–·1′–·1′ (·1, ·1)–(·1, ·1)–·15′–(·15, ·1 (·15, ·15)–(·15, ·15); $_1$ (·2, ·2)–(·2, ·2)–(·2, ·2)– (·25, ·25)–(·25′, ·25)–3; ($_2$·3, ·3)–(·3, ·3)–(·3, ·3)–·3 $_1$·3; $_2$·3. Still under tre ment

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Dec. 12th, 1891	Absolute rest in bed. Antiseptic dressings. Subsequently plaster jacket and a month in a convalescent home	Note on April 6th, 1891.—"Patient still wearing the Sayre's jacket. The sinuses remain soundly healed, and there is no pain in the back. General condition of patient is good" *Result.*—Healing of the sinuses. Disappearance of pain (In a similar case I would now employ Koch's treatment. There can be no question that it was of great benefit to the patient in this instance)	The patient suffered very little at the commencement of the treatment. After the first injection the only symptoms were headache and pain in the back and general malaise. On Dec. 19th it is noted that the sinus in the back had almost healed, and that in front was looking much more healthy. On Dec. 30th both the posterior wounds were soundly healed, and a probe could not be passed into the anterior wound for more than 1 inch. On Jan. 7th there was a small scab over this wound, and when that was picked off a probe could not be introduced. When the dressing was changed on Feb. 8th this wound was found soundly healed, and she had then no pain or tenderness over the spine. A Sayre's jacket was applied on Feb. 17th, and she was sent to the convalescent home on Feb. 20th to have 1 injected every 3 days. She returned at the end of March and went back to her school, arranging to attend for injection every other day. Remains well in July.
Injections commenced on Feb. 10th. Total number of injections up to April 8th, 1891 = 60. Total amount up to April 8th, 1891 = 7·709 grms.	None	Note on April 4th, 1891.—"Sinus still open, but much retracted and smaller. No thickening in front of joint. Inguinal glands still large on both sides and more distinct. Glands in neck in *statu quo*" *Result.*—Improvement in so far that the bare bone has become covered and the sinus is become drawn in (This is a case where I now think that this treatment should be of value, and in which I would use it. There seems little reason to doubt that so far advantage has been gained by it)	In the first three reactions the temperature went above 104°, and there was cough, sickness, and headache. After the first injection the hip swelled up and became tender, but this quickly subsided. On March 3rd it is noted that most of the thickening about the hip has disappeared, there is very little discharge; the sinus still persisted, and the orifice was level with the surface of the skin. Treatment stopped in May. Not yet healed.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
30	J. P., æt. 8, male, K. C. H., admitted Feb. 10th, 1891. *Diagnosis.*—Recurrent disease of knee after arthrectomy; septic sinus rendered aseptic	One brother has spinal disease, no other tubercular family history. Has suffered from disease of his left knee-joint for 5 years; has been in various hospitals, and was admitted into my ward in Oct., 1889. On Oct. 16th of that year I performed a partial arthrectomy, removing all the diseased synovial membrane in front of the joint but not dividing the ligaments to get at the back. The wound healed by first intention, and on Nov. 6th the leg was put up in plaster of Paris and the child sent home. For some months the joint went on very well, but he was readmitted at the end of July, 1890, with recurrence of the disease. Complete arthrectomy was then performed. The wound healed by first intention, and the patient was sent out wearing a plaster of Paris case. In October it was found that a small abscess had formed over the external condyle	Internal organs healthy. Two sinuses on the outer side of the left knee-joint; otherwise the part appears healthy. On Feb. 13th these sinuses were connected, and the cavity which went down to the outer side of the femur thoroughly cleared out, wound left open (The above is the treatment which I would have adopted before the Koch period)	·002; $_1$·003; $_1$·005–·01″ $_1$·01″; $_1$·01″; $_2$·01″; $_1$·01″ ·01″–·01′–·15′–·02′–·02–·03– ·04′–·04′–·015–·02; $_1$·03–·04 ·05′; $_2$·06–·07–·08–·09′–·1–·1 (·1, ·1)–(·1, ·1)–(·1, ·1)– (·1, ·1′)–(·1, ·1)–(·1, ·1)– (·1, ·1)–(·1, ·1)–(·1, ·1)– (·1, ·1)–(·1, ·1). To continu
31	F. R., æt. 19, female, K. C. H., admitted Feb. 14th, 1891 *Diagnosis.*—Disease of wrist-joint with septic sinus rendered aseptic	Tubercular family history. Disease of left wrist began 15 months ago. No known cause. Abscess opened 4 months ago	No lung trouble. Some pus in urine and irritability of bladder. Disease of left wrist-joint. Thickening around joint, especially over lower end of radius. Great pain on the slightest movement, septic sinus towards the inner side of the back of the wrist leading into the joint and to bone. On Feb. 24th the wrist-joint was excised by the long posterior incision, the sinus being enlarged and also utilised. The wound was sponged out with undiluted carbolic acid, the long incision stitched up and a drainage-tube put in where the sinus was. The long incision healed by first intention, and the sepsis had evidently been eradicated. The drainage-tube was still in when the injections were begun (Before the Koch period I would have excised the wrist-joint in this case)	·002‴; $_2$·002′; $_1$·003 $_1$·003″–·003′–·003–(·003, ·0 (·004, ·003)–(·005, ·005)– (·008, ·008)–(·01, ·01)– (·01, ·02)–(·02, ·02)–(·02, ·0 (·03, ·03)

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Feb. 20th, 1891. Total number of injections up to April 8th, 1891 = 49. Total amount of fluid used up to the same date = 3·1 grms.	Antiseptic dressings	Note on April 5th.—"Dressing changed to-day after a week's interval. A point the size of a pin's head not yet quite healed" *Result.*—Rapid healing of the wound (This is the sort of case in which I think Koch's treatment is of value) April 20th.—The sinus was found healed at the next dressing	The first 3 injections produced no local or general effect. The subsequent ones caused headache, cough, and pain in the left knee. There is nothing further to note; the wound gradually filled up and at the last note was almost completely healed. Treatment stopped in May. Remains well.
Injections commenced on March 20th, 1891. Number of injections up to April 8th = 24. Amount of fluid used up to the same date = ·257 grms.	Usual treatment after excision of wrist	Note on April 8th, 1891.—"Some œdema around wrist, long wound apparently soundly healed, small wound looking healthy and closing rapidly, very little discharge" *Result.*—Still under treatment; improving so far; too early to judge (According to my present views the treatment adopted in this case promises a better result than excision alone) April 20th.—Wounds completely healed; no thickening	The patient was not particularly ill after the injections. There was a good deal of local reaction, which still continues. There was no increase in the pus in the urine. Drainage-tube left out on April 5th. Sent home in May, quite healed.

We had six cases where there were *aseptic* sinuses in connection with disease of bones and joints. Of these, one (No. 32) has only been under treatment for a very short time. Of the remaining five, two (Nos. 27 and 28) have completely and apparently soundly healed, and have so far derived the greatest advantage from the treatment; while one (No. 30) is almost absolutely healed, and I think will be found quite healed when the dressing is next changed. In one case (No. 29) healing is not yet complete, but the condition is steadily improving, and the bare bone which was present in the first instance can no longer be felt. In one case (No. 26), so far as the bone is concerned, no marked benefit has been derived, though bare bone felt at first has now disappeared; in this instance the delay in healing is, I think, due to the conditions of the sinus itself rather than to the presence of tubercular disease. In this patient a tubercular sore on the hand healed under treatment, but has relapsed to some extent since the treatment was discontinued. (Note on April 20th.—Of these six cases only two (Nos. 26 and 29) now remain unhealed.)

C.—CASES WITH SEPTIC SINUSES.

c. *Cases wit*

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
32	B. S., æt. 19, female *Diagnosis.*—Disease of spine with septic sinus; disease of elbow with septic sinus; disease of occipital bone	No tubercular family history. In Feb., 1889, abscess formed in leg, and pain in back was complained of. In commencement of 1890 abscess formed in connection with two ribs, and were opened. In March a psoas abscess was opened in the left lumbar region. In July signs of left elbow-joint disease commenced, and an abscess was opened in Oct. In Nov. swelling and pain were noticed over the occipital bone	Patient anæmic and weak. Sinuses in connection with ribs, spine and elbow. Large fluctuating tender swelling over occipital bone. Lungs and other organs healthy. No hectic temperature (Before the Koch period I would have simply provided free escape for discharge in the first instance)	·005‴; ·007‴; ·007‴ ₄·007‴; ₄·008‴; ₃·008″ ₄·008″; ₄·008″; ₄·008′ ₂·008.″ Treatment discontinued
33	A. E. K., æt. 22½, male, K. C. H., admitted Nov. 29th, 1890. Discharged March 24th, 1891 *Diagnosis.*—Tubercular disease of the right os calcis and the astragalo-calcanean joint with septic sinus	No tubercular family history. When about 9 months old swelling first noticed on outer side of right ankle. Has been treated at various hospitals since. An abscess formed, and broke some months ago	Internal organs healthy. Marked swelling about the right heel and ankle, especially on the outer side. A septic sinus is present below and behind the external malleolus, and leads directly inwards to bare and soft bone, apparently the upper surface of the os calcis and the astragalo-calcanean articulation. No loose bone can be felt (Before the Koch period I would have cleared out the diseased bone)	·0023‴; ₂·0023″: ₁·003′ ₂·003″; ₁·003″; ₁·003 ₂·003′; ₁·003′; ₂·004 ₁·004′; ₄·005″; ₂·005′ ₁·005′; ₁·005; ₂·006′; ₁·007 ₁·008′; ₂·008″; ₁·008 ₁·01′; ₂·012″; ₁·015; ₁·0 ₂·03″; ₁·03: ₁·04′ (oper tion, interval of 13 day ₁·01′; ₂·02′; ₁·02′; ₁·0 ₁·03′; ₂·03; ₁·04′; ₂·0 ₁·06″; ₁·07; ₂·07″; ₁·0 ₁·08′; ₂·09″; ₁·1″. Tre ment stopped on Mar 20th, 1891

eptic Sinuses.

'otal number, nount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Injections ommenced on v. 28th, 1890; discontinued Jan. 2nd, 1891. nder treatment for 35 days. tal number of jections = 10. otal amount of fluid used = ·074 grms.	None beyond the ordinary dressings, rest in bed, and splint for the arm	Last seen on April 3rd, 1891. Patient much thinner and somewhat weaker than in November. All former sinuses still open, and an additional sinus leading to the occipital bone, which is bare. Tenderness and small fluctuating swelling over upper part of sternum. No hectic temperature; lungs and other organs healthy. Patient's general condition is improving, but has still occasional retching *Result.*—Improvement during treatment as regards the unopened swelling over the occipital bone; spinal condition unaltered; condition of elbow and general state of patient worse (I would not now employ the Koch treatment in such a case. It was a hopeless case from the first)	After the first injections patient suffered much from malaise and sickness. The elbow became more painful, but the swelling over the occipital bone after the initial increase steadily diminished, and almost disappeared before the treatment was stopped. Towards the end of Dec. the retching and incapacity to take food became worse, and the pain in the elbow increased. Cultivations were made from the sinuses in the loin and elbow, and staphylococcus cereus albus was obtained. On Jan. 6th, 1891, the elbow was freely opened, and a quantity of tubercular material scraped out. After the anæsthetic she had continuous sickness for several days, which greatly reduced her. On account of her feeble condition and the possibility that the retching before the operation might have been connected with the treatment and the presence of the pyogenic organisms, it was deemed unadvisable to resume it after the operation. The patient slowly recovered from the operation, and the swelling over the occipital bone re-formed, and was incised on Feb. 12th. At the end of Jan. pain and swelling were observed over the upper part of the sternum on the right side, and some cheesy material was evacuated at the beginning of March. Died in May.
Commenced c. 3rd, 1890. Treatment stopped on March 20th, 91, lasted for 107 days. al number of ections = 41. otal amount of fluid sed = 1·0146 grms.	On Feb. 3rd the opening was enlarged and a loose sequestrum was found and removed, the cavity from which it was taken being scraped and sponged with undiluted carbolic acid. Wound left open. On March 23rd it was again scraped out	Note on April 6th, 1891.—"Cavity in os calcis with free external opening. No bare bone, wound looks healthy. Less swelling" *Result.*—No apparent effect on the disease (I cannot see that the patient has obtained any advantage from the Koch treatment; in a similar case I should now follow the old lines and only use tuberculine if the wound failed to heal and if I had got it aseptic)	Not seriously ill after the injections, but especially after the 4th, 5th, and 6th much cough. After the 1st injection the heel on the outer side became red and much swollen, and there was greatly increased discharge. The thickening diminished considerably, but as bare bone was still felt, the sinus was opened up on Feb. 3rd. The wound did not heal, and it was again thoroughly scraped out on March 23rd.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount o injections.
34	H. A., æt. 21, male, K. C H., admitted Dec. 1st, 1890. Discharged on Feb. 16th, 1891 *Diagnosis.*— Disease of elbow-joint with septic sinuses	Tubercular family history. The disease in his left elbow began 14 months ago without apparent cause. At first there was only a feeling of weakness in the elbow, and some swelling. Then 11 months ago, when opening a window, had pain which rapidly increased with increase of swelling. Abscesses soon formed, and were opened from time to time, and the joint was thoroughly cleared out after division of the olecranon last August by Mr. Clutton. The sinuses did not heal	Internal organs healthy. Right elbow-joint much swollen, the swelling being of a soft pulpy character, with numerous septic sinuses leading into the joint and to soft carious bone; the whole appearance of the part is that typical of a bad case of tubercular disease with septic sinuses. No movement is possible. The elbow is held at a little beyond the right angle. The circumference of the joint at the level of the tip of the olecranon is $1\frac{7}{8}$ in. larger than on the left side	·01‴; $_1$·01′; $_1$·015′; $_4$·015 $_1$·015′; $_2$·02; $_1$·025; $_3$·025 $_4$·03″; $_2$·03′; $_1$·04′; $_1$·0 $_2$·08″; $_3$·06; $_2$·08′; $_1$· (operation interval of days); $_1$·04; $_1$·06′; $_2$·08 $_1$·08; $_1$·1; $_2$·1; $_1$·12; $_1$ Discharged, and twice week had ·1. Treatme stopped at the end of Mar
35	E. P., æt. 3 years, female, P. G. C. H., admitted Dec. 3rd, 1890. Discharged on Mar. 10th, 1891 *Diagnosis.*— Disease of radius with septic sinus; enlarged cervical glands	No phthisical family history. The disease of the radius began a year ago after injury. Has been in a general hospital and operated on without benefit	Enlarged glands in neck. Internal organs healthy. Septic sinus at back of lower end of right radius leading upwards to bare bone; no loose fragment felt. End of radius considerably thickened (The treatment in such a case before the Koch treatment would have been operation with the view of removing the affected portions of bone)	·002‴; $_5$·002‴; $_4$·002′ $_1$·002″; $_1$·002″; $_1$·002 $_2$·002″; $_1$·002″; $_7$·002 $_2$·002′; $_4$·004″; $_1$·00 $_1$·005″; $_2$·005; $_1$·00 $_1$·007′; $_2$·008; $_1$·01; $_1$·00 $_2$·012; $_1$·015; $_1$·02 (operati 12 days' interval); $_1$·01 $_1$·015–·02′–·02′–·02–·02–·03 $_1$·03′–·03–·04–·05–·06″; $_2$·0 ·07; $_1$·08′–·1′–·12; $_2$·15–·1 $_1$·15. Treatment disc tinued
36	E. M., æt. 26, female, K. C. H., admitted Dec. 16th, 1890, discharged on Feb. 23rd to be treated at home *Diagnosis.*— Disease of fibula with septic sinuses	Phthisical family history. Six years ago had disease of right thumb, from which bone was removed. This was followed by abscesses in the forearm. Four years ago abscesses formed in connection with the lower end of the fibula, and sinuses have remained since from which small pieces of dead bone have lately come out	Internal organs healthy. Scars on right forearm and thumb, the last phalanx of which has evidently been almost entirely destroyed. About 2 inches above the external malleolus are 2 septic sinuses leading to bare and soft bone about 1 inch in extent. There is thickening of the right tibia towards its upper part, but this is not at all painful (Before the Koch period I would have laid open the sinuses and thoroughly scraped and gouged away the diseased bone, but I think it is not at all improbable that the removal of the affected piece of the fibula would have been ultimately necessary in order to effect a cure	·01‴; $_4$·008‴ (interval o days); ·008‴; $_2$·008 $_2$·008″; $_1$·008″; $_1$·008 $_2$·01′; $_1$·012′; $_1$·012′; $_2$·01 $_1$·015′; $_1$·02′; $_2$·03′; $_1$·0 ·05–·05; $_1$·06′; $_1$·06′; $_1$· $_2$·07′; $_1$·08′; $_2$·08′; $_1$· $_4$·09′; $_1$09; $_1$·09; $_4$·1; $_1$· $_3$·1; $_1$·1. $_1$·1; $_2$·1; $_3$·1

tal number, ount, &c., of njections.	Additional treatment.	Condition when last seen.	Notes.
lections com- ced Dec. 3rd,); stopped at d of March. al number of tions = about 34. tal amount of fluid ed = about ·275 grms.	On Dec. 9th the sinuses were opened up and drainage-tubes inserted; the joint was not scraped out. On Jan. 23rd the sinuses were again opened up and the joint thoroughly scraped out; the tubercular material came away very readily	Note on April 6th, 1891.—"All the sinuses are still open, and there is no very marked improvement in the condition of the joint as compared with its state before treatment was commenced" *Result.*—No noticeable improvement (In such a case I would not now use Koch's treatment)	The first reaction was not very severe, but the elbow swelled up very much, and there was increased discharge from the sinuses. During the course of the treatment several small acute abscesses formed, and were opened from time to time. The tissues around the elbow became much firmer, and the measurements less. The patient was discharged on February 16th to be treated as an out-patient; at that time the wounds were granulating well, and, with the exception of one in front of the elbow, were looking well and healing up rapidly. Bare bone could only be felt at one part. Excised in May.
ommenced . 9th, 1890. l number of ctions = 42. l amount of used = 1·349 grms.	On Feb. 5th the sinus was enlarged and a loose sequestrum was found and removed, undiluted carbolic acid applied, wound left open.	Still a small short sinus at one part of the scar which does not go to bare bone. General health good. Glands smaller than at first *Result.*—During the treatment the sequestrum became loose: the wound is healing up well. Glands smaller (In such a case I would now operate in the first instance, and if I got the wound aseptic, and if it would not heal advantage might be obtained from this treatment. Possibly some advantage may have been got here from the previous use of the treatment, in that the separation of the sequestrum may have been favoured)	The first 2 reactions were severe, the chief symptoms being coughing, swelling of glands in neck, pallor, and sickness. There was swelling and increased discharge from the wound. After the operation the wound gradually closed but has not yet quite healed.
mmenced 28th, 1890. nber of injections up to il 5th, 1891 = 34. l amount of used up to at date ·772 grms.	The sinuses were opened up on Jan. 15th, and a counter opening was made behind. Bare bone was felt. Tubes inserted	Note on April 6th.—"All the sinuses are still open, but are very small and drawn in and there is very little discharge. No bare bone can be felt" *Result.*—Sinuses closing up. Bone no longer bare (In a similar case I would now lay open the sinuses, scrape the bone, try to render the wound aseptic and commence Koch's treatment at once. I think that in this case the treatment was decidedly beneficial in leading to the removal of the carious material at the surface of the bone)	The first reaction was not very severe; there was a good deal of local reaction and the discharge was profuse and very offensive. A small piece of bone came away on Dec. 31st. She suffered from diarrhœa after the first 2 injections and was slightly jaundiced. Nothing further noteworthy as regards reactions. On Feb. 1st no bare bone could be felt, and when she was sent home to be treated by Dr. Brown the sinuses were healing rapidly; the tubes had been left out some days previously.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount injections.
37	F. M., æt. 10, male, K. C. H., admitted Dec. 29th, 1890, discharged Feb. 5th, 1891, to attend as out-patient *Diagnosis.*—Recurrent disease after excision of elbow-joint; septic sinus	Disease of elbow joint, commenced when 3 years old. After an injury and an abscess formed soon afterwards. Has been operated on twice by Mr. Wood, and a year ago the joint was excised by Mr. Barrow. At Mr. Barrow's request the patient was submitted to the Koch treatment as a sinus still remained unhealed, and as there was a fresh development of tubercular disease in connection with the internal condyle	Internal organs healthy. Sinus in bend of elbow not leading to bone. Over the internal condyle is a soft semifluctuating mass with the skin red over it and a small opening. Movements of joint fair (Before the Koch period I would have thoroughly cleared out the mass over the internal condyle and scraped out the sinus)	·003″; $_{1}$·003′; $_{1}$·0 $_{2}$·004″; $_{1}$·004′; $_{1}$005′–· $_{1}$·008′; $_{1}$·01; $_{1}$·015; $_{2}$ $_{1}$·03; $_{1}$·04; $_{2}$·05′; $_{1}$·05; $_{2}$·06; $_{3}$·07; $_{2}$·08; $_{5}$·09. attended once or twi week since the 18th of J and had ·1. Treatr stopped on April 8th

otal number, ount, &c., of injections.	Advanced treatment.	Condition when last seen.	Notes.
Commenced c. 31st, 1890. tal number of njections = about 34 tal amount of luid used = ut 2·002 grms.	None	Note on April 8th.—"The sinuses still exist and show no tendency to heal. The condition being practically the same as before the treatment" *Result.*—No improvement (I would now treat such a case in the same manner as before the Koch treatment, and if it seemed necessary afterwards, that treatment could be employed in addition)	After the first injection a good deal of fluid was discharged from the swelling over the internal condyle. The thickening soon disappeared and only a small sinus was left. After the 2nd injection the patient developed a papulo-vesicular rash in patches on the sides and back exactly in the lines and presenting the superficial appearance of herpes zoster. This gradually died away in about a fortnight. There is nothing further to note.

There were six cases of diseases of bones and joints with *septic* sinuses. Of these four cannot be said to have derived any benefit; one (No. 36) undoubtedly has, and one (No. 37) is healing after operation, and possibly the separation of the sequestrum has been expedited by the treatment. In No. 32 it is interesting to note that the tubercular deposit where the skin was unbroken improved under treatment, but that those with septic sinuses did not; indeed, one, the elbow, became worse. Add here Case No. 1, where there were septic sinuses in connection with a metacarpal bone, and where the condition improved markedly under treatment, having healed in the first instance.

DISEASES OF GLANDS.

III.—DISEA

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount injections.
38	P. O. F., æt. 30, male, K. C. H., admitted Nov. 30th, 1890. Discharged Jan. 29th, 1891 *Diagnosis.*—Tubercular glands in neck and axilla with abscesses	Sister died of phthisis. Glands in neck began to enlarge 6 years ago, and those in axilla 1 year ago. Have been increasing much during the last year. Medicinal treatment	Internal organs healthy. There is a large mass of enlarged glands matted together and situated in the right anterior triangle at the upper part and beneath the sterno-mastoid; there were two small abscesses on the surface of this mass, a large number of hard but separate glands along border of sterno-mastoid and in posterior triangle, quite visible. On the left side of the neck there were also a number of large glands, especially at the upper part of the anterior triangle, but they were not so large as to cause deformity. In the right axilla there was a large mass of glands matted together and projecting at the anterior and inner border of the axilla. At the back of the left wrist there is a fluctuating swelling evidently connected with the sheaths of the tendons (Before the Koch period I would have recommended excision of the glands)	·01‴; $_2$·006″; $_3$·00 $_3$·006″; $_2$·006″; $_1$·0 $_1$·007″; $_1$·008′; $_4$·00 $_2$·008″; $_1$·008″; $_1$·008′–· $_1$·015; $_1$·02′; $_1$·03′; $_2$· (operation, interval of days); $_1$·03″; ·03″; $_1$·0 $_2$·04′. Discharged to go with treatment at ho (He only had 4 injection home)
39	L. B., æt. 12, female, P. G. C. H., admitted Dec. 6th, 1890. Discharged to attend as out-patient on Jan. 23rd, 1891 *Diagnosis.*—Tubercular cervical glands	Doubtful phthisical family history. Enlarged glands in neck for a year. No evident cause. Corneal nebula on right side	Internal organs healthy. Enlarged glands along posterior border of both sterno-mastoids. On right side opposite middle of sterno-mastoid there is one gland hard and of the size of a Brazil nut; the others are about the size of beans: upper lip swollen and fissured (Before the Koch period I would have left these glands alone, giving a *placebo* such as cod-liver oil, &c.)	·005″; $_1$·004″; $_1$·0 $_1$·005″; $_2$·005″; $_1$·0 $_1$·007′; $_1$·009′; $_2$· $_1$·012″; $_1$·012; $_2$·015; $_1$· $_2$·025; $_3$·03; $_2$·035; $_1$ $_1$·04; $_2$·05; $_1$·06′; $_1$ $_2$·06; $_3$·06; $_3$·06; $_4$ $_2$·06; $_5$·07. Treatr stopped

OF GLANDS.

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Dec. 3rd. 1890. Total number of injections = 25. Total amount of fluid used = about ·489 grms.	On Jan. 16th the abscesses in the neck were scraped out; they were quite superficial, and only contained a small quantity of cheesy material. The mass in the axilla was excised, and found to consist of a number of large, almost completely caseous glands. The wound healed by first intention. The glands were in the main cheesy, but portions were found containing tubercles. Two guinea-pigs were inoculated with portions of the glands on Jan. 16th, and became tubercular	When discharged on Jan. 29th the wounds were completely healed. The mass in the neck was about ⅓ of its original size, and the individual glands of which it was composed could be readily felt. The glands on the left side were also distinctly smaller. The swelling on the wrist remained the same In a note dated April 7th, 1891, Dr. Garrod Thomas says: "I examined him to-night and find that the glands originally affected (right side) are smaller, but there are several fresh ones considerably involved just above the clavicle on the left side; the wrist and armpit are quite well" *Result.*—Improvement in that the glands became somewhat smaller and much less matted together (In such a case I would still recommend excision, but it is quite a question whether it would not be advantageous to precede the operation by a few injections. Certainly the operation would be very much easier, safer, and more satisfactory if the periadenitis were in this way got rid of)	The first reaction was a severe one, the chief symptoms being headache, vomiting, and rigor. An erythematous rash appeared on his chest about 10 hours after the injection, and faded in a few hours. There was considerable local swelling, and one of the abscesses in the neck burst and discharged a little pus. During the next few reactions the rash and rigors generally recurred, and he had a good deal of nausea. The mass in the neck diminished gradually in size, and the individual glands could be made out a few days after the treatment was commenced.
Commenced ec. 9th, 1890. Stopped b. 16th, 1890. eatment continued for 69 days. No. of injections = 27. otal amount of fluid d = ·824 grms.	None	When the treatment was stopped the glands were distinctly smaller than they were originally Note on April 6th.—"Glands much smaller than at last note. The lip is well" *Result.*—Distinct diminution in the size of the glands. Apparently no tendency to relapse (I would not now trouble to use the Koch treatment in such a case)	Nothing noteworthy about the reactions except that the upper lip swelled after the first injection. The patient did not suffer from the treatment at all, but after the 4th injection she was noticed to be slightly jaundiced; no other symptoms; this passed off in 2 or 3 days. As the glands were almost stationary and small, and there were no reactions, the treatment was stopped.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
40	G. G., æt. 2, male, K. C. H., admitted June, 1890. Discharged Jan. 26th, 1891 *Diagnosis.*—Tubercular disease of inguinal and cervical glands	No phthisical family history nor history of syphilis. Last summer patient was operated on for hydrocele of the cord and afterwards for phymosis. The patient was sent home on the same day that circumcision was performed. When seen some days later the circumcision wound presented the appearance of a tubercular ulcer. The scar from the hydrocele operation broke down shortly afterwards, and presented the same appearance. The testicle became tubercular and was removed in autumn. Glands in both groins became tubercular and broke down. The child rapidly wasted	Large ulcers in both groins, with greyish sloughy surfaces. Enlarged gland on left side of neck. Child much emaciated; abdomen large and tense. No lung symptoms. Hectic temperature. As the parents were extremely anxious that this treatment should be tried, their wish was acceded to	·001′; 3 ·002″; 1 ·0023′; 1 ·003″; 1 ·004″; 1 ·005–·006; 1 ·007; 1 ·008′; 1 ·01″ 8 ·006″; 4 ·008; 2 ·01′; 1 ·01″; 1 ·01″; 15 ·005; 3 ·005
41	E. C., æt. 5, female, P. G. C. H., admitted Dec. 8th, 1890, discharged Jan. 26th, 1891. *Diagnosis.*—Tubercular glands; sinus leading to diseased bone	Tubercular family history. Six months ago was in Infirmary with disease of left fibula. Some bone was removed by operation. An abscess formed in lumbar region lately and has burst	Internal organs healthy. Scar about 3 inches long adherent to the lower end of the fibula. Bone enlarged at upper end of scar. There is a large mass of enlarged glands in the right iliac fossa, and a sinus joint above the right posterior superior iliac spine leading to bare bone at upper part of crest of ilium. No signs of vertebral or sacroiliac disease	·002″; 1 .003″; 1 ·003‴; 1 ·002‴; 2 ·0025″; 1 ·003″; 1 ·004″; 1 .004″; 2 ·004; 1 ·005; 1 ·007; 2 ·01; 1 ·015; 2 ·02′; 1 ·025′; 1 ·03′; 2 ·035; 1 ·04; 1 ·04′; 2 ·05; 1 ·06; 1 ·06′. Sent to convalescent home and ·06 given twice a week
42	A. A., æt. 7, female, P. G. C. H., admitted Sept. 22nd, 1890 *Diagnosis.*—Tubercular glands with septic sinuses in groins; tubercular glands in neck; slight phthisis	Has attended the hospital for 2½ years with suppurating tubercular glands in both groins, and has been admitted and operated on several times. Sinus opened and scraped on Sept 25th, and caseous material removed from left iliac fossa. The remains of the glands on the right side were cut out	Enlarged glands in neck. Small area of crepitation at angle of left scapula. Liver enlarged, occasional attacks of diarrhœa. Scars of old sinuses in both groins, and a sinus in each groin discharging pus. On both sides there is thickening in the iliac fossa, most marked on the left; eyes and ears normal. Hectic temperature	·003‴ 1 ·003″; 1 ·003‴; 1 ·002‴; 2 ·003″; 1 ·003″; ·003″; 1 ·004; 2 ·005; 1 ·005′. Treatment abandoned

Total number, mount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced on Nov. 26th; last jection on Jan. th. Length of treatment 62 ays. Number injections = 17. tal amount of id used = ·1023 grms.	None	Died at home about the end of February. Parents did not inform us of the death till a fortnight later, and therefore there was no post-mortem examination. One of the glands in the neck had rather increased *Result.*—No noticeable effect as regards groin; increase in one cervical gland (I would not now employ the Koch treatment in such a case)	There were no general effects after the injections beyond the elevation of temperature. No distinct local reaction except in the cervical glands. After the first injections the temperature in the intervals became normal instead of hectic, and the child seemed much brighter and took food better. The injections were given up on Jan. 4th, but a fortnight later the hectic temperature returned and the child began to go downhill rapidly; hence the resumption of the injections. As, however, the parents wished to take the child home, they were allowed to do so. When discharged the local condition was very much the same as when the treatment was begun, but the child was thinner and weaker.
Commenced ec. 9th, 1890. tal number of jections up to ril 8th, 1891 = 42 tal amount of id used up to he same date 1·6245 grms.	Sent to convalescent home on Jan. 26th, 1891	Note on April 7th.—"Condition the same as when sent to the convalescent home *Result.*—Some diminution of the mass in the iliac fossa; sinus *in statu quo* (In such a case I would not now employ the Koch treatment)	No noteworthy general symptoms. At first there was increased swelling in the iliac fossa, and increased discharge from the sinus. The swelling in the iliac fossa soon diminished, and when the child went to the convalescent home on Jan. 26th it was much smaller than it was originally. A small piece of dead bone had come away, but bare bone could still be felt. There was no local reaction in the fibula.
menced Dec. , 1890. Left on Dec. 30th, 0. Continued or 21 days. tal number of injections = 10. tal amount of 1 used = ·034 grms.	Excision of hip after the treatment was stopped. See notes. The joint was much inflamed; there was evidently an acute septic arthritis	Note on April 6th, 1891.—"Wounds healed, except where drainage-tubes are. A good deal of purulent discharge. Hectic temperature. Child anæmic and losing flesh. Condition of lungs unaltered" *Result.*—Extension of septic process under treatment. No improvement as regards tubercle (According to my present views, I would not now employ the treatment in such a case. There seems little reason to doubt that harm was done here by the treatment in that the extension of the septic process (not of the tubercle) was favoured)	After the 1st injection there was much local reaction in left groin. The discharge from the sinuses afterwards diminished, and for a time the condition of the patient was satisfactory. On Dec. 31st symptoms of disease of the left hip-joint commenced, and the discharge from the sinuses was much more profuse. As the temperature remained high after the last injection, the treatment was abandoned. On Jan. 27th the left hip-joint was excised, the sinuses were opened up, and a counter opening made in the left lumbar region. The hectic temperature has continued, though not very marked.

To these we must add thirteen others where enlarged glands were present, but which are mentioned under other headings. In seven of these (Nos. 3, 11, 12, 15, 22, 53, and 54) no perceptible difference can be made out in the size of the glands; in four (Nos. 8, 18, 35, and 45) it is noticed that they were somewhat smaller; in one the glands were looser but not smaller (No. 29); and in one (No. 23) they became smaller under treatment, but one or two of them enlarged after the treatment was stopped. In five cases the enlargement of the glands was the principal or only apparent affection, and in three of these (Nos. 38, 39, and 41) there was no external communication. In all three the glands diminished somewhat in size, and in one (No. 38) where they were at first much matted together the periadenitis disappeared in a very striking manner. Of two cases where there were sinuses in connection with the glands, in one no noticeable effect was produced (No. 43), while in the other (No. 42), whatever may have happened to the glands themselves, the septic suppuration spread in the neighbouring tissues to a very serious extent.

TUBERCULAR PHTHISIS, ETC.

IV.—Tubercula

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
43	W. P., æt. 11, male, P. G. C. H., admitted Dec. 8th, 1890, discharged to attend as out-patient on Feb. 14th, 1891 *Diagnosis.*—Tubercular peritonitis	Tubercular family history. Was admitted into hospital under Mr. Boyd in May, 1890, with signs of tubercular peritonitis and a history of wasting of 3 months' duration. On May 31st Mr. Boyd opened the abdomen and found a tubercular mass in the sub-peritoneal tissue, matted intestines studded with miliary tubercles, and 2 pints of turbid fluid in peritoneal cavity. Hard masses were also felt in the pelvis. Cavity washed out with warm water, and 2 drachms of iodoform and glycerine emulsion injected. Wound stitched up and healed by first intention. Patient sent to convalescent home in July much improved. Readmitted in October, the scar having opened at the lower part. A quantity of caseous material was removed from the subperitoneal tissue. This wound healed, but broke down again about the end of November	A few crepitations at right apex in front. No other physical signs. Has occasional attacks of abdominal pain, headache and vomiting. Abdomen not distended, skin not inelastic, and no ascites. Slight dulness over hypogastric and iliac regions. On deep pressure hard masses felt in neighbourhood of umbilicus and scar. In middle of scar is small sinus covered with a scab. Small ulcer on outer side of left buttock. Weight 3st. 8½ lbs. (Before the Koch period I would simply have continued the antiseptic dressings)	·003‴; ₂·003‴; ₁·003‴ ₁·003‴; ₂·003‴; ₁·003″ ₁·003″; ₁·003′; ₂·004′ ₁·005; ₁·007″; ₂·009′; ₁·012 ₄·015; ₁·02″; ₂·02′; ₁·02‴ ₆·015′ ₁·015; ₂·02; ₁·025 ₁·03; ₂·03; ₁·03; ₁·04′ ₁·05–·05; ₂·06; ₂·06; ₁·06′ ₁·07; ₂·07; ₁·07′; ₁·07; ₂·07 ₁·09; ₁·1; and ·1 once week afterwards

V.—Tubercular Iri

44	E. B., æt. 9, female, Moorfields Ophthalmic Hospital, under the care of Mr. Waren Tay *Diagnosis.*—Tubercular iritis	No definite tubercular family history. Speck on iris noticed for three weeks. No complaint of pain, but eye inflamed. No evidence or history of syphilis	A few enlarged glands in neck. No disease found in internal organs. "Slight occasional shooting pains in right eye. A good deal of circumcorneal congestion. Aqueous, turbid. Iris, light cocoa brown (left iris bluish grey), many posterior synechiæ. Four small rounded white deposits near lower papillary margin Marked keratitis punctata. T + (?) Vision is bare perception of bright light"	·001″; ₁·001′; ₁·002′ ₁·002‴; ₁002‴; ₁·002 ₄·002; ₅·004′; ₂·005; ₄·00

Peritonitis.

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Dec. 8th, 1890. Total number of injections up to April 8th, 1891 = 41. Total amount of fluid used up to same date = 1·661 grms.	None	Note on April 6th, 1891.—"Wound healed. Patient apparently in good health. No pain or swelling in the abdomen" *Result.*—Improved; sinus healed; thickening in pelvis subsided (It is probable that this is one of the cases in which advantage will be gained by the Koch treatment)	The first reactions were severe, the chief trouble being sickness. There was also a little temporary albuminuria. On Jan. 17th the reaction after ·02 was again severe, there being sickness and diarrhœa, the latter persisting for several days accompanied with abdominal pain; several of the motions contained blood. There was no marked increase of discharge from the sinus which had healed by Dec. 23rd. The weight went down to 3st. 5¾ lb. on Dec. 28th, and remained till Jan. 26th. On Feb. 5th it was 3 st. 7 lbs.

and Corneal Ulcers.

Commenced Dec. 3rd, 1890. Last injection on Dec. 31st. Under treatment for 28 days. Total number of injections = 10. Total amount of fluid used = ·028 grms.	Eye excised on Jan. 7th, 1891. On examination numerous tubercles were found in the iris. Two rabbits and one guinea pig were inoculated on Jan. 7th with portions of the iris, and became tubercular	The patient has not been seen since she left the hospital at the end of Jan. *Result.*—No apparent effect. Continued progression of the disease	Although the temperature after the fourth and fifth injections was above 104° the child was not particularly ill. There was never any distinct local reaction. On Dec. 8th it was noted that the "cornea is steamy. More bulging in ciliary region, especially below. The white nodules are becoming confluent, and there is a large swelling at the outer part." On Dec. 23rd, after an interval of five days, it was evident that fresh tubercles were appearing; indeed the whole iris had become mottled white, and the original tubercles were merged into the swollen iris and hardly distinguishable. As the condition got worse, the eye was excised on Jan. 7th, 1891. On Jan. 12th ·009° was injected, and a distinct reaction occurred, the temperature reaching 101·5° eighteen hours after the injection, but no evidence of local reaction could be found anywhere. On Jan. 7th portions of the iris were introduced into the eyes of two rabbits, and into the subcutaneous tissue of the abdomen of a guinea pig. Three weeks later there was distinct tubercular iritis in both rabbits, and in the guinea pig there was a nodule at the seat of inoculation, and markedly enlarged inguinal glands on that side.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
45	L. P., æt. 10, female, P. G. C. H., admitted Dec. 31st, 1890. *Diagnosis.*—Corneal ulcers; enlarged glands in neck	Intense photophobia and discharge from both eyes for 8 months. For last 6 months has only opened eyes in the dark. Treatment.—Boracic lotion.	Mr. Jessop's notes.—"In both eyes follicular conjunctivitis. On right eye central corneal nebula, apparently no active ulceration going on. No iritis. On left eye an active ulcer below centre of cornea not very deep." Intense photophobia. Weight on Jan. 16th 2 st. 11½ lbs. Enlarged glands in neck	·002‴; $_3$·002‴; $_4$·002′; ·002‴; $_2$·002‴; $_1$·002″; $_1$·003″; $_1$·003′; $_1$·0035″; $_2$·0035′; $_1$·005′–·007′–·009″; $_1$·01–·01′–·01′–·01′–·01′–(·01′, ·01′); ·015′–(·015′, ·02′); $_1$(·02, ·02)–·03″–·03′; $_1$·03′–·045′–·16′–·08′–·1′–·12; $_1$·15″–·15–·2′–·25–·25″–·25′; $_1$·25′;–·25″–·25′–·3′; $_1$·2′; $_1$·3′–·3′ Treatment discontinued on March 25th

In connection with this case see also No. 22, where ther

VI. TUBERCULAR LARYNGITIS.

46. C. W., æt. 28, tubercular laryngitis without any evident phthisis, under the care of Dr. Greville Macdonald. I regret that as Dr. Macdonald happened to be ill when these notes were being compiled, I cannot give the details of the case. Speaking from memory, however, I may say that the patient had been under treatment for some months with tubercular ulceration of the larynx (bacilli present), and the greater part had healed; a small portion of the ulcer, however, was not easily accessible, and remained stationary. The treatment was begun at the commencement of December, ·001 being given at first with only very slight local or general reaction. Four more doses were given at that time, the last being ·01, but withou producing any general febrile reaction, though locally there seemed to be some increased redness and swelling. The treatment was then given up for some weeks, but as no improvement occurred, it was resumed after about 5

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
ommenced Jan. 15th. Total number of injections = 46. Total amount of fluid used = 3·801 grms.	Cod liver oil. Boracic lotion. Sent to convalescent home on March 25th, 1891	Note on April 7th.—"The local and general condition have continued to improve since the patient left the hospital. There is hardly any photophobia; no congestion; no ulceration. Small nebulæ on each cornea. Cervical glands rather smaller" *Result.*—Healing of the ulcers; gradual cessation of the photophobia; slight diminution in size of glands	During the reactions there was sickness and headache, but no marked increase in the eye symptoms. On Jan. 22nd Mr. Jessop notes that there is active ulceration in the right corneal nebula; photophobia and slight discharge from the eyes. On 29th he notes less congestion on the right side, and ulcer still active on left. On Feb. 5th he notes eyes improved. On Feb. 19th he states, "Left eye, conjunctiva much congested and slight muco-purulent discharge, corneal ulcer healing up, and less photophobia. In right eye conjunctiva congested, no active corneal mischief, nebula as before." The condition has steadily improved. On Feb. 20th weight 2 st. 12½ lbs. There was no loss of weight during the early treatment.

as a corneal ulcer which healed quickly under treatment.

weeks, the first dose given then being ·01. This was followed by severe general and local reaction. The next dose was ·004 without reaction, and in all 10 injections were given without reaction after the first. During this treatment, however, fresh ulceration appeared in the neighbourhood and on the epiglottis, and the treatment was, whether wisely or not I cannot say, given up. I have no doubt, however, that we made a mistake in throwing aside the treatment at first, and in another like case, instead of abandoning the treat ment on the second occasion, I should be inclined to reduce the dose and employ the "continuous" method of administration.

To this case may be added 3 other cases which will be found in the list of phthisis cases. In one (No. 48) only a few injections were given, and no change was noticed in the laryngeal condition. In the other two (Nos. 49 and 50) the ulceration healed, and has not, so far as I know, broken down again. The total result, then, is 4 cases, 2 healed, 1 *in statu quo*, and 1 worse.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
47	Mrs. M., æt. 39, under the care of Dr. Waterhouse. *Diagnosis.*—Phthisis	Tubercular family history. General health good until 4 years ago when she was nursing her sister who was dying of phthisis at Mentone, and was much exposed during the earthquake. She came home with cough and numerous tubercle bacilli in sputum. Since then she has wintered abroad, and the disease has only progressed slowly till last summer, when it began to extend more rapidly	Patient rapidly losing flesh; profuse night sweats. Hectic temperature (evening temperatures 102° and 103°). Going down hill very rapidly and not expected to live more than a couple of months. As the patient and her friends begged to have a trial of the treatment it was agreed to give her a few injections to see if any good was likely to be done. There was a large cavity at the left apex with dulness and crepitation involving the upper half of the lung. Dulness and crepitation at the right apex but no definite signs of a cavity	·002‴; $_1$·002″; $_1$·003″ $_2$·0035″; $_1$·004″; $_1$·005″ $_1$·006″; $_1$·008″; $_1$·008″ $_2$·008″. Treatment stopped (Most of these reactions ar marked moderate, but as matter of fact the evenin temperatures before th treatment was commence ranged about 103°, and i a considerable number o instances after the injection the highest temperature wa 102°, and the other sym ptoms of reaction were for th most part absent)
48	Dr. B., æt. 27, medical man,, under the care of Dr. G. A. Sutherland *Diagnosis.*—Phthisis and tubercular laryngitis	Tubercular family history. Six months ago symptoms of lung disease began, and have been getting rapidly worse since September, when slight hectic fever set in, loss of appetite, wasting and weakness. His voice first became husky in August	Suffers from frequent cough with expectoration (about 5 oz. daily), nummular, bacilli abundant. At right apex consolidation, with bronchial breathing, and below the clavicle impaired resonance and occasional crepitations. Cavity (?) immediately below clavicle. Dulness with weak breathing and crepitations at right base posteriorly. Left lung apparently normal. Larynx examined by Dr. Greville Macdonald, who found slight ulceration on posterior wall of larynx, with congestion and swelling of both cords. Other organs healthy. Weight 10 st. 3 lbs. Patient complains of complete loss of appetite and great weakness	·002‴; $_1$·002″–·003″–·008″ $_1$·004″; $_1$·004″–·005′–·007′– ·007′; $_1$·01′; $_1$·012′; $_1$·015′ $_1$·015′; $_1$·02′–·02″; $_1$·02′ $_1$·025′; $_2$·03′; $_1$·035′; $_1$·05″ $_2$·05′; $_1$·06′; $_1$·08′; $_2$·1″; $_2$·1′ $_3$·1; $_2$·12; $_3$·15′; $_2$·1; $_3$·1 $_2$·1; $_3$·1; $_2$·1; $_1$·1; $_1$·1; $_1$·1 $_2$·1; $_1$·1; $_1$·1; $_1$·1; $_1$·1; $_1$·1 $_1$·1; $_1$·1; $_1$·1. Left for Ne Zealand on March 11th. T continue ·1 every day every second day accordin to circumstances
49	Mrs. H., æt. 40, under the care of Dr. G. A. Sutherland *Diagnosis.*—Phthisis and tubercular laryngitis.	No tubercular family history. The commencement of the disease dates from July, 1889, and the symptoms lately have been cough, profuse expectoration, increasing weakness and loss of flesh, huskiness, diarrhœa alternating with constipation, fever, and profuse night sweats. No hæmoptysis	Advanced disease of both lungs, right chiefly affected with a large cavity below the clavicle; no cavity on left side. Ulceration affecting the posterior part of the right vocal cord and the posterior wall of the larynx. Hectic temperature; evening temperature about 102°; profuse nummular expectoration with numerous bacilli	·0015; $_1$·002; $_1$·003–·003 $_1$·004; $_1$·005–·007; $_2$·007 $_1$·007; $_1$·008

PHTHISIS.

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Nov. 27th, 1890. Last injection on Dec. 16th, 1890. Under treatment for 19 days. Number of injections = 10. Total amount of fluid used = ·0495 grms.	None	Died on Feb. 10th, 1891. No post-mortem examination permitted *Result.—In statu quo*	The chief symptoms during the reactions were increase of cough and difficulty of breathing. The treatment was stopped because it did not seem to be doing any good and the patient was getting weaker. There was no evidence that the treatment had done any harm. When it was stopped on Dec. 16th there was no alteration in the physical signs, the night sweats had stopped and the temperature at night was lower than before the treatment, and continued so for some weeks; the patient however was becoming progressively weaker. After the first injection a patch of herpes appeared on the right trochanter and afterwards developed into a bedsore, as she lay constantly on that side.
Commenced Nov. 28th, 1890. Number of injections up to March 9th = 45. Total amount of fluid used up to that date = 2·749 grms.	None	Note on March 9th.—"Occasional cough in the morning. About ½ an ounce of mucus dotted with pus expectorated during the 24 hours. Extremely few bacilli. The only physical signs are consolidation at right apex with slight bronchial breathing and no crepitations nor signs of cavity. Over the rest of right lung there is slightly impaired resonance; breathing rather faint, vesicular. Left lung apparently normal. Larynx normal. Weight 10 st. 7½ lbs. (Patient states that 10 st. 7 lbs. is his normal weight.) General condition excellent" *Result.*—Great improvement	After the first injection some moist sounds were also noticed at the left apex behind, otherwise no marked increase in the lung symptoms. Improvement was noted on Dec. 3rd, and steadily progressed with the exception of slight hæmoptysis on Feb. 9th, which did not recur. After the first injection the voice was huskier and there was acute œdema of the posterior wall of the larynx. On Jan. 6th Dr. Macdonald reported that the ulceration had quite healed, and it has remained well since. The bacilli became much fewer, but have not yet entirely disappeared. His general condition rapidly improved; he regained his appetite and strength, fever disappeared, and he was able to walk long distances without fatigue. Patient lost 2 lbs. during the first few weeks, but has lately been gaining weight. He left England on March 11th as surgeon to a steamer sailing to New Zealand Returned in July. Larynx remains well. Lungs very much improved. Still continuing the treatment.
Commenced Nov. 28th, 1890; discontinued Dec. 14th, 1890. Under treatment 16 days. Number of injections =10 Total amount of fluid =·0475 grms.	None	Died of hæmoptysis about a month after the treatment was discontinued. No post-mortem examination, as the patient had left London *Result.—In statu quo*	As regards the reactions, the only sign was slight increase over the normal hectic temperature. There was no alteration in the lung or laryngeal symptoms. The treatment was only commenced at the very earnest request of the patient and her relatives, and as it had no apparent effect on her condition, it was discontinued on Dec. 14th.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
50	W. R. S., æt. 27, medical man, under the care of Dr. Guthrie *Diagnosis.*—Phthisis and tubercular laryngitis	Family history. One brother had necrosis of tarsus. Patient had influenza in Feb., 1890, followed by general bronchitis. Eustachian catarrh, and otitis media, with perforation of both tympana. Cough severe. Night sweats and wasting. Severe attacks of hæmoptysis in August and November, 1890, and acute laryngitis. Signs of early consolidation at left apex, noted after hæmoptysis	Anæmic. Weight 11 st. 4 lbs.; has been gaining lately. No hectic or night sweats. Constant paroxysmal cough with much expectoration. ℥v—℥x daily of frothy mucus containing little solid matter (bacilli found a few days later). Very deaf. Both tympanic membranes gone. Voice weak and hoarse. Cords much congested; do not meet on phonation. Superficial ulceration of posterior part of left vocal cord (Dr. Macdonald). At left apex dulness in Sup. Sc. F. and resonance diminished to 3rd rib in front and mid-scapula behind. Expansion deficient. Breath sounds weak. Inspiration cogged; expiration prolonged. Faintly bronchial in Sup. Sc. F. Faint moist râles over this area. Breath sounds somewhat harsh and expiration prolonged over right lung, but no accompaniments	·001; ·002′–003′–004″; $_{1}$·005′; $_{1}$·006′–008′–·01″; $_{1}$·01′; $_{1}$·012; $_{1}$·015′; $_{1}$·02′; $_{1}$·02″; $_{1}$·02′; $_{1}$·02′; $_{1}$·025′; $_{1}$·03′; $_{2}$·04′; $_{1}$·05; $_{1}$·07′; $_{2}$·1′; $_{1}$·12; $_{1}$·15; $_{2}$·2″; $_{2}$·15′; $_{3}$·15′; $_{2}$·15′; $_{3}$·11′; $_{2}$·1′; $_{3}$·11′; $_{2}$·11; $_{4}$·12′; $_{1}$·1; $_{2}$·12; $_{3}$·12; $_{3}$·14′; $_{2}$·14; $_{3}$·12; $_{1}$·1; $_{4}$·1. Left as surgeon on board a steamer to continue injections every 3 days or oftener if possible
51	Mrs. F., æt. 29, under the care of Dr. Guthrie, went home on Jan. 29th, 1891 *Diagnosis.*—Phthisis	Symptoms of phthisis for 2 years. Tuberculosis on mother's side. 4½ years ago, laryngitis (aphonia) congestion of vocal cords). 3 years ago, abscess in great toe; right metatarso - phalangeal joint. 16 months ago, dry pleurisy left apex. 12 months ago, wasting, and cough. Yellow lumpy phlegm. Never hæmoptysis. Profuse night sweats 9 months ago, cavity at left apex formed	Complains only of cough. Expectoration ℥j—℥iij daily, purulent nummulated. Contains bacilli about 5 to the field. No night sweats for last 6 months. No hectic. Spare and delicate looking. Finger tips clubbed. Slight œdema over shins. Weight 8 st. 4 lbs.; used to weigh 9 st. At left apex cavity size of Tanjerine orange. Deficient expansion. Dulness from lower border of 2nd left rib in front to middle line of scapula. Tympanitic resonance in first left space. "Cracked pot" sound. Amphoric breath sounds with moist râles at end of inspiration but not abundant. Loud whispering pectoriloquy all over dull area, and increased vocal resonance. Right chest. Harsh breath sounds at inner part of 2nd space, no accompaniments. Larynx.—Showed congestion (slight) of vocal cords. No ulceration. Heart.—Normal. Urine.—Normal	·002″; $_{1}$·002′–·003′–·004″ $_{1}$·005″–·006′–·008′; $_{1}$·01′; $_{1}$·01′; $_{1}$·01′–·012′; ·015″–·015‴; $_{3}$·015″; $_{3}$·02″; $_{1}$·03′ $_{2}$·05′; $_{4}$·05″; $_{1}$·05′–·065′ $_{1}$·08′; $_{2}$·1‴; $_{5}$·08″; $_{3}$·08′ $_{1}$·08′; $_{1}$·1′. Went home on Jan. 29th to continue injections of ·1 twice a week At the end of March she was advised to continue the injections and increase their frequency

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Treatment commenced on Nov. 28th. Total number of injections up to March 9th =40 Total amount of fluid used up to that date =2·881 grms.	Cod-liver oil and hypophosphites	Note on February 6th.—"Considerable improvement. Weight fell from 11 st. 4 lbs. to 10 st. 11¼ lbs. during first fortnight of injections. Present weight 11 st. 2 lbs. Much more colour in cheeks; general health good; a few bacilli still present in sputum. Amount of sputum varies from 1½ to 3 oz., and is very watery. *Chest.*—Resonance distinctly better below left clavicle; still impaired, but not absolutely dull in left Sup. Sc. F. Air entry good; expiration prolonged; a few crackling râles above clavicle and in Sup. Sc. F. *Larynx.*—Slight congestion. Ulcer on vocal cord not quite cicatrised" In the middle of March he wrote: "I feel perfectly strong, and except for a very occasional cough, hoarseness, and tendency to cold feet, am all right. No bacilli in sputum last week. Two examinations, and no dulness anywhere in the chest or back" *Result.*—Improved, and improvement progressing	Reactions never severe, attended usually by increase of cough, and on two occasions by headache, gastric pain, and vomiting. Pain and tenderness over left side of larynx was complained of on December 22nd, and lasted a few days. Signs of slight dilatation of right bronchus were noted on Dec. 11th, with tenderness on percussion, and a few pleural frictions on 2nd right space. Pleurisy soon subsided. Neither voice nor hearing improved. Went home to continue injections at home on Feb. 7th, 1891. Left in the middle of March as surgeon on board a vessel bound for the West Coast of Africa.
Commenced, Nov. 8th, 1890. Total number of injections up to end of March = about 4. Total amount of fluid used up to same date = about 2·707 grms.	Cod-liver oil and hypophosphites	On April 6th Dr. G. E. Williamson reports, "I examined Mrs. F. a week ago. Her general condition is much improved, she looks much stronger, eats and sleeps well. Her breathing on going uphill is also improved. The expectoration is about ¾ oz. in the 24 hours, nummular and yellow. The chief local improvement in the lungs is the almost complete absence of moist sounds" *Result.*—Improved, and improvement progressing	Reactions severe only on 2 occasions, after ·015 and ·1 respectively. Similar doses repeated caused no malaise. Signs of right apical catarrh and patch of consolidation under right scapula size of shilling appeared during 3rd week of inoculation and persisted. Signs of slight right apical pleurisy noted about same time, which soon disappeared. On Jan. 28th Dr. Guthrie reports, "Dulness slightly less at left apex below clavicle. Very few moist râles. No apparent change in size of cavity. Still a few moist crepitations at right apex, and small patch of consolidation beneath right scapula persisted. General condition highly satisfactory; gained 2 lbs. during inoculation period. Can walk 8 to 9 miles without fatigue. Expectoration less, ʒj—ʒiss per diem, still purulent and nummulated, and a few bacilli present." On March 18th she wrote that the cough was very distinctly better. At end of June still steadily improving. Treatment being continued.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
52	F. W. F., æt. 29, male, medical man, under the care of Dr. Frederick Taylor *Diagnosis.*—Phthisis	No tubercular family history. In Oct., 1881, had an attack of hæmoptysis, lasting 3 days, but not profuse; no previous symptoms and no physical signs. Since then has had 6 attacks; the last 8 months ago. No physical signs discovered till last July, when patient developed somewhat acutely physical signs of tubercle at the left apex associated with much pyrexia. Was ill for 2 or 3 weeks, and then till November was pretty well, but had cough and expectoration. This became much worse in Nov., accompanied with loss of appetite, loss of flesh, had night sweats and much increased cough and expectoration	Nov. 29.—Pale and thin with much cough, and with tremor on exertion; expectoration of mucopus; night sweats; weight, 10st. 12oz. Examination of chest (with Dr. Goodhart). Slightly impaired movement under left clavicle; very slight impairment of resonance above clavicle, on clavicle and in first space; slightly deficient vesicular murmur, and increased expiratory murmur above clavicle. Crackling râles above clavicle, and below down to level of nipple. Behind the same râles from apex over scapula to midway between spine and lower angle (7in. from 7th cervical spine). Slightly increased vocal resonance at left apex. Right side, apex healthy. Occasional sibilus below clavicle	·002‴; $_2$·002″–·002″–·003″ ·004″–·004′; $_1$·005″; $_1$·005″ $_1$·005″; $_1$·005′; $_1$·006′ $_1$·007‴; $_1$·007″; $_1$·006′ $_1$·005″; $_1$·006′; $_1$·007″ $_4$·008″; $_1$·008′; $_1$·01′ $_2$·012′; $_2$·012′; $_1$(?)‴; $_4$·01′ $_1$·012′; $_1$·014″; $_3$·012″ $_3$·012; $_1$·015′; $_3$·02′; $_2$·025′ $_1$·035″; $_1$·035′; $_3$·04′; $_5$·03′ $_1$·04″; $_1$·04′; $_2$·04′; $_1$·045′ $_2$·05′; $_1$·045–·05′–·05‴ $_2$·04′; $_1$·04; $_1$·04. To go o with injections every day (every second day, graduall raising the dose
53	E. R., æt. 9, female, P. G. C. H., admitted Dec. 5th, 1890, under the care of Dr. Herringham. Discharged to attend as out-patient on Jan. 24th, 1891 *Diagnosis.*—Phthisis; enlarged cervical glands	Tubercular family history. Wasting and cough for nearly 2 years. When first seen in Jan., 1890, she complained of cough and sweating at night, and there were signs of consolidation and breaking-down at right apex. This condition continued much the same till admission, her weight being 2 lbs. less than when first seen	A few enlarged glands in sub-submaxillary region. Slightly impaired resonance at both apices, with some crepitation on the left side. No sputum could be obtained for examination. No night sweats or diarrhœa. Other organs normal. Weight 3 st.	·002″; $_1$·002″; $_1$·003′ $_1$·004″; $_2$·004′; $_1$·005 $_1$·007′; ·009″; $_2$·01; $_1$·015′ $_1$·02′; $_2$·03″; $_1$·03′; $_2$·04 $_1$·05′; $_1$·05; $_2$·05′; $_1$·06 $_1$·06; $_2$·06′; $_1$·06; $_1$·0 $_2$·06; $_3$·07; $_2$·07; $_3$·0 $_2$·07; $_3$·07; $_2$·07; $_3$·0 $_1$·07; $_2$·07; $_1$·07 (6 day interval ·09 and ·1 once week (4 subsequently)
54	E. M., æt. 8½, female, P. G. C. H., admitted Dec. 8th, 1890, under Dr. Sidney Phillips. Discharged Jan. 26th, 1891. Readmitted Mar. 11th *Diagnosis.*—Phthisis and enlarged glands in neck	No phthisical family history. Subject to winter cough. Four montha before admission, severe cough commenced with expectoration; loss of weight and night sweats	Enlarged glands in neck, various organs except lung healthy. Consolidation at base of right lung reaching as high as angle of scapula posteriorly, and 3rd rib anteriorly, with bronchial breathing and fine and coarse crepitations. No sputum could be got for examination. Night sweats not marked. Weight 2 st. 8 lbs. Temperature normal	·001″; $_1$·001′; $_1$·002 $_4$·002‴; $_1$·002; $_1$·004′ $_1$·004′; $_1$·005″; $_1$·00 $_1$·006; $_2$·008; $_1$·01; $_2$·01 $_1$·02; $_1$·025; $_2$·03′; $_1$·03 $_1$·04; $_2$·05′; $_1$·06; 1 Sent to convalescent hom and had ·06 twice a wee After readmission of Mar 11th (·06, ·07)–·1″–1′–15; $_1$·2′–·25″; $_1$·3′; $_1$3′–3′–3 ($_1$·3, ·3)–·3; ($_2$·3, ·3)–3′–3′ ·3′; ·3; ·3

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Dec. 1st, 1891. Total number of injections up to March 21st, 1891 = 46. Total amount of fluid used up to that date = about ·9 grms.	None	March 21, 1891 (interval of 16 weeks). Feels and looks much better than in November. Has very little cough, practically no expectoration. Sleeps fairly well and rarely sweats at night (did so on 15th, but only slightly). Appetite good. Weight, 10st. 3½lb. Examination of chest:—Left side, impairment of resonance scarcely to be noticed; breath sounds deficient; crackling râles above clavicle, and less marked down to nipple level, but as compared with state on Nov. 29th have cleared up over costal cartilages (inner half of area); whisper audible above clavicle. Behind left side, râles from apex down to nearly same point as on Nov. 29th, *i.e.*, 6in. from 7th spine, but confined to inner half. Right side, breathing supplementary above and below clavicle; no râles. Increased vocal resonance and audible whisper above clavicle *Result.*—Great improvement in general health, cough, expectoration, sweating, physical vigour, slight increase in weight. Physical signs, diminished extent on left side. Very slight early indications at right apex (clicks have been heard on 2 occasions)	The first reactions were somewhat severe, but there was no evident increase in the pulmonary signs. On Jan. 16th I injected by mistake a decigramme instead of a centigramme which I had intended. I at once incised the swelling and also injected some absolute alcohol to precipitate the tuberculine, and thus no doubt a good deal of the fluid was got rid of; the subsequent reaction was about as severe as the first reaction. On March 13th, after a second dose of ·05, the patient had also a pretty severe reaction, apparently a cumulative effect; the only instance of the kind which I have observed at such a late period in a case. This patient also went on reacting much longer than any other that I have had, as shown by the fact that after 46 injections I had only got up to 4 centigrammes. He left on March 23rd as surgeon on board a vessel bound for the Cape of Good Hope to continue the treatment for some months. Seen in June. General health good. Condition of lungs slightly improved. Did not continue injections after leaving England.
Commenced Dec. 9th, 1890. Number of injections up to April 4th, 1891 = 38. Total amount of fluid used up to that date = 1·881	None	On April 6th Dr. Herringhamnotes: "The local condition is, I think, exactly the same as when the treatment was begun; the general health is rather improved." Condition of glands about the same *Result.*—Slight improvement	No noticeable increase in the lung symptoms occurred after the first injections. Improvement noticed at the end of December, and when sent home the improvement was very marked, the percussion note at left apex being normal and no crepitations present. At right apex a few râles were occasionally heard on deep inspiration, and there also the percussion note was normal.
Commenced December 9th. Total number of injections up to April 8th =54. Total amount of fluid used up to same date =6·459 grms.	Sent to convalescent home on Jan. 26th	Note on April 6th.—"Her present condition is the same as on Jan. 26th. No general symptoms, and patient is apparently in good health. Glands *in statu quo*" *Result.*—Marked improvement	Nothing to note about the reactions. During the course of the treatment the crepitations diminished considerably, and the consolidation became much less marked with the exception of the base of the lung anteriorly. When sent to the convalescent home on Jan. 26th the condition was as above, there being left only a small area of dulness around and below the right nipple with occasional crepitations. Very slight cough. No night sweats when in hospital. At the home the disease seemed to relapse somewhat, and she was therefore readmitted and the treatment pushed. Since that was done improvement has again taken place. The weights were on Feb. 8th, 2 st. 8 lbs.; Feb. 16th, 2 st. 11½ lbs.; Feb. 28th, 2 st. 11 lbs.; Jan. 9th, 2 st. 11½ lbs.; Jan. 17th, 2 st. 11¼ lbs; Jan. 26th, 2 st. 12½ lbs.; March 2nd, 2 st. 13 lbs.

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
55	J. H. S., æt. 30, medical man, under the care of Dr. Leslie Ogilvie *Diagnosis*,—Phthisis	No history of phthisis in the family. Been active and well till about the beginning of December, when he felt tired and overworked. Had hæmoptysis on Dec. 30th, which continued for about a week. Bacilli tuberculosis found in sputum about Jan. 4th in large numbers	*Personal appearance.*—General nutrition and muscular development deficient. Thin face; high cheek bones. Clear delicate complexion with slight hectic flush. *Symptoms.*—Debility. Short cough with mucous expectoration, though not abundant, and containing numerous tubercle bacilli. *Physical signs.*—Flat chest, deficient expansion of right apex. Percussion note fairly resonant. Inspiratory murmur feeble. No crepitations heard. Vocal resonance and fremitus normal. Fauces congested. Larynx normal. Pulse 80, regular and full. Evening elevation of temperature (about 100° F.) Weight on Jan 12th = 8 st. 9 lbs.	·002″; $_2$·002′–·003″; $_1$·003 $_1$·003″; $_1$·003′; $_1$·00 $_1$·005′; $_1$·007′; $_1$·00 $_1$·01″; $_1$·01; $_1$·015′; ·0 $_1$·025′; $_1$·04′; $_2$·05′; $_1$·0 $_1$·07′; $_1$·08; $_1$·09; $_1$·1; Went home, ·1 to be injec every other day for a ti and then every day

Thus there were nine cases of phthisis, of which two were hopeless from the first (Nos. 47 and 49), and in which no change was noted in the lungs during the short injection period. Of the remaining seven cases all improved in general condition, some of them very markedly so; as

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Injections commenced on Jan. 14th, 1891. Total number of injections up to April 8th = about 38. Total amount of fluid used up to that date = about 2·21 grms.	None.	At the commencement of March it is noted.—"General appearance improved. No cough or expectoration. No crepitations heard. Respiratory sounds normal. A gland on the left side of the neck, which became prominent during the treatment, is smaller, but is still enlarged slightly. No bacilli when sputum last obtained for examination. Pains which were felt after each injection in the left knee and elbow are no longer noticed. Weight on 28th of Feb. = 8 st. 13 lbs." In a note on March 14th he says that expectoration has now entirely ceased, and that his weight was 9 st.; and on March 30th he writes that his lungs were examined 2 days previously and that nothing abnormal cou'd be detected. His weight was then 9 st. 1¼ lbs., and he felt better able to bear fatigue *Result.*—Improved, and improvement continuing	Nothing special to note about the reactions. After two injections, auscultation disclosed crepitation and harsh inspiration over an area between 2nd and 4th ribs in front towards axillary border on right side. This continued for about 10 days. Complained a good deal of pain in left knee and elbow after the early injections. Patient went home at the beginning of March and resumed his practice.

regards local condition in one (No. 53), it is said to be the same as at first, while in the others there has been improvement, varying from "slight" to "very remarkable." To these we must add No. 24 (improved) and No. 42 (*in statu quo*).

VIII.—Injections in Non-Tubercular or Doubtful

interesting, and may

No.	Name, age, sex, &c.	History.	Condition before injection.	Frequency and amount of injections.
56	S. M., æt. 54, female, K. C. H., admitted Dec. 9th, 1890 *Diagnosis.*—Anæsthetic leprosy	Born in Germany. Was a nurse in Calcutta for 27 years, and had cases of leprosy under her care. Came to England 10 years ago. The disease began in 1880 with pain and stiffness in joints and a macular rash over abdomen; shooting pains in limbs. Shortly afterwards the body was covered with dark red raised painless blotches, first noticed about the head, each lasting for a year to a year and a half. In 1881 hands and feet swelled, pieces of bone came from ends of fingers and toes, and nails dropped off. At the same time she complained of numbness and feeling of weight in limbs. Deformity of the fingers has come on since, but for the last 7 years the disease has been practically quiescent. In September last first complained of photophobia, lachrymation and pain in the right eye	Internal organs apparently healthy. Anæsthesia not complete anywhere, but sensation is very imperfect as high as the middle of the legs and forearms, and somewhat impaired higher up. Pain on pressure on calves especially on right side, no definite enlargement of nerves; possibly the right ulnar behind the internal condyle is a little thicker than the left. Fingers flexed into palm; cannot be extended; cannot oppose thumb. No anæsthetic patches over body	$\cdot01'''$; $\cdot01''$; $_2\cdot01''''$; $_3\cdot005'''$; $_2\cdot002'$; $_3\cdot004'$; $_5\cdot004'$; $_1\cdot005'$; $_1\cdot006$; $_2\cdot008$–$\cdot01$; $_3\cdot012$; $_1\cdot015$; $_1\cdot015$; $_2\cdot02$; $_3\cdot03$; $_2\cdot05$; $_1\cdot06$; $_1\cdot08$; $_2\cdot1$; $_1\cdot1$–1; $_2\cdot1$; $_1\cdot1$; $_2\cdot1$. Injections discontinued

57. E. S., æt. 23, female, was admitted on account of a little pain in her knee. This disappeared in a few days with massage. A centigramme was injected, and to our surprise there was considerable general reaction, the temperature reaching 103·4° 27 hours after the injection. There was no reaction in the knee at all, but the patient showed us 2 scars on the right arm which had resulted from chronic abscesses and which had reacted. She told us that they were not quite well before the injection, but they gave her no trouble, and she had forgotten to mention them.

58. A student at the hospital with a sinus on trochanter, not supposed to be tubercular. 1 centigramme produced no effect.

59. A. F., a woman with an undoubted rheumatic knee, did not react to 1 centigramme.

60. J. D., æt. 31. A case of typical lupus erythematosus, did not react after 1 centigramme.

61. W. S., æt. 5. Post-nasal growths, did not react after 2, 4, and 6 milligrammes.

62. H. R., æt. 6. Post-nasal growths, did not react after 2 and 4 milligrammes.

63. J. F., æt. 15 months. Syphilitic epiphysitis of lower end of tibia, did not react after 2 milligrammes.

64. E. P., æt. 7. Inflammation of hip after acute rheumatism, did not react after 2, 4, and 8 milligrammes.

65. A case of syphilitic disease of the nose, palate, pharynx, &c., in a boy, did not react after 2 milligrammes, but after 1 centigramme there was a

Cases. (The first, a case of leprosy, is very
be given in detail.)

Total number, amount, &c., of injections.	Additional treatment.	Condition when last seen.	Notes.
Commenced Dec. 10th, 1890. Discontinued on Feb. 18th, 1891. Length of treatment = 70 days. Total number of injections = 27. Total amount of fluid used = 1·156 grms.	None	Report from Dr. Walter C. S. Burney, Superintendent of the Greenwich Union Infirmary, dated April 4th, 1891.—"She states that before she was injected she suffered with pains in the extremities every month, lasting from 4 to 5 days. These pains have not since reappeared. The anæsthesia of the arms existed up to the junction of the middle and upper third of the radius whereas it now does not reach higher than about the middle of the metacarpal bones; about the same may be said of the lower extremities. She has increased in weight and this cannot be attributed to any extra diet. She complains that she has not been able to get her breath so well since the injections; she has had one or two slight fainting fits. On the whole I am of opinion that she has greatly improved, and the patient herself attaches great importance to the relief of the periodic pains" *Result.*—Improvement in some respects	The first reaction was severe, the chief symptoms being rigor, vomiting and oppression of breathing. There were shooting pains in arms and legs, a large vesicle formed on the inner side of the left great toe, and there was swelling of the left fore and middle finger. Next day a macular rash in the form of a line was seen on the lower folds of the mammæ meeting in the centre. Another line across the upper part of the abdomen and a semi circular patch below and on the right of the ensiform cartilage. There was also another line at the posterior edge of the right buttock. After the 3rd injection the patient was collapsed in the evening, when the temperature 6 hours after the injection was 97°, pulse 44 and respirations 40 and very shallow. The symptoms subsided as the temperature rose; the highest temperature was 103°. Fresh bullæ appeared on the left leg and on the right heel and ankle. There was no further trouble, the rash faded between each injection and came out again during the reactions; it had not entirely disappeared when she left the hospital. After the 16th injection patient asserted that she could open her hand better than before the treatment and that she herself felt better; the anæsthesia was somewhat less marked. When she left the hospital she still asserted that she was better in these respects.

general febrile reaction (temperature above 102°) but no local reaction whatever.

66. A. B., female, æt. 49, was admitted with a scar at the left angle of the mouth with several ulcerating patches on it. Patient had suffered from ulceration here for 23 years, and a great variety of diagnoses have been made, among others that of lupus. It seemed most probably a case of rodent ulcer, but a series of injections were given commencing with ·002 grammes. She had general febrile reactions, not severe, after the first six injections, but we never could satisfy ourselves as to any local reaction. Not the slightest alteration occurred in the disease during treatment.

67. Mrs. W., a nurse under the care of Dr. Greville MacDonald with masses of soft tissue in both nostrils, as to the nature of which many diverse opinions have been expressed by those who have made clinical or microscopical examinations, some thinking that it must be some form of tubercular disease. In her case a number of injections were given, beginning with ·002 and going as high as ·3, and towards the end of the treatment the part was scraped from time to time. There were distinct febrile reactions from the first, and the patient said that there was increase in the nasal discharge, but the local reaction was never very definite. Whether the improvement which followed the treatment was entirely due to the scraping, or to it and the tuberculine combined, and whether the disease was tubercular or not, are points which are not yet clear. For my own part I have always been sceptical of its tubercular nature, and I cannot find any evidence of tubercle in the portions which I have examined microscopically.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 124.)

THIRTEEN CASES

OF

PAGET'S DISEASE OF THE NIPPLE,

WITH

SPECIAL REFERENCE TO THE CAUSATION OF THE MALADY BY PSOROSPERMS.

BY

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Received February 10th—Read May 12th, 1891.

It is now fifteen years since specimens of Paget's disease of the nipple were first shown at a meeting of the Royal Medical and Chirurgical Society by Mr. Butlin, and on subsequent occasions this surgeon, Mr. Henry Morris, and Dr. Thin have given details of additional cases. For the past ten years, however, the subject has not been again brought before this Society, although illustrative cases have been published elsewhere; and as it is one which has recently attracted considerable attention, I have thought the present a favorable time to again bring the matter forward. I am the more disposed to ask

the attention of Fellows because when this disease was last discussed there was some difference of opinion as to its pathology, and the nature of the morbid processes involved; and because, although much additional information has been obtained of late years, the total number of recorded cases is yet small, and the records of additional examples may prove of value.

For the following thirteen cases I am indebted to the Surgical Staff of St. Bartholomew's Hospital, where most of the patients have been under treatment, and to my friends Mr. Everley Taylor of Scarborough, and Dr. Stowers and Dr. E. S. Tait of London. In all cases the whole of the parts removed have been placed in my hands for purposes of examination, and in almost all of them I have had the opportunity of examining the patients before operation.

Case 1.—J. B—, æt. 53, came under the care of my friend Mr. Everley Taylor, of Scarborough, in March, 1882. She was a married woman, but had never borne children, and had had no trouble with her breasts. Three years before coming under notice a chronic irritation of the nipple and areola commenced, which she thought was caused by the friction of her stays. Eighteen months later this began to spread more quickly and to supply more exudation than formerly. After a further interval of six months she first noticed a lump in the breast.

On examination the nipple was found to be much retracted, and was surrounded by a raw, red, excoriated surface covered with thin scales. Beneath the nipple and continuous with it was an irregular, hard, and lumpy tumour. The axilla was free.

On March 6th the breast was amputated, and on section was found to be occupied by a typical scirrhous growth. The patient made a good recovery, and five years later remained quite well.

Case 2.—M. B—, æt. 36, was admitted into St. Bartho-

lomew's Hospital under the care of Mr. Cripps on May 20th, 1882. She was a married woman and had borne five children. She had suffered from cracked nipples whilst suckling, but these had always healed after weaning the children. Otherwise she had enjoyed good health, and came of a healthy family, but her mother's sister had died of cancer of the stomach.

Her history was that a year before admission, and without apparent cause, her right nipple became cracked and sore, and never healed in spite of treatment. Six months later she noticed a lump in the breast.

On admission it was found that the right nipple was retracted and excoriated. Around it for a distance of three quarters of an inch was a ring of bright red ulceration. The edge of this ulcer was not sharply defined, but faded into eczematous skin with crusts and scales. Above the nipple, but situated in the upper part of the breast close to its edge, was a hard nodular growth the size of a chestnut, and in the axilla was one enlarged and hard gland. On May 24th the breast and the enlarged gland were removed. A section of the former showed a typical scirrhous carcinoma at the upper margin of the breast, and separated from the nipple by apparently healthy tissue. The gland also was cancerous.

Case 3.—E. W—, æt. 52, a married woman, came under the care of Dr. Stowers in July, 1882.

Her left breast had been removed for cancer three years previously, and the right nipple had been sore and excoriated for two years, and had steadily got worse.

The right nipple and areola were a bright red. At Dr. Stowers' request I removed the nipple and about half an inch of the adjacent skin for microscopical purposes, and then Dr. Stowers destroyed the remainder of the affected area with Vienna paste. After that time I lost sight of the patient for a year and a half, at the end of which period she came to St. Bartholomew's Hospital, and remained under treatment by Mr. Cripps for some time.

She was then suffering from an enormous fungating tumour situated over the sternum, and apparently extending through that bone from the mediastinum. The right breast was quite healthy, and the scar left by the Vienna paste was quite sound. The operation scar in the left breast was also sound.

Case 4.--H. K— was admitted into St. Bartholomew's Hospital on May 1st, 1883.

She was a healthy woman, æt. 45. She had had four children, and since the birth of the third child, seven years previously, the left nipple had been sore. The last child refused to take the breast, and for the past two years the soreness of the nipple had spread over the areola, and had caused her a good deal of burning pain. Four months before admission the patient first noticed a tumour in the lower part of the left breast at some distance from the nipple. She had previously enjoyed good health, and there was no family history of cancer.

An examination of the breast showed that the nipple and the areola were bright red and congested. The epidermis was partly destroyed, and a red, raw surface was exposed exuding some blood-stained serous fluid. In the lower part of the breast and quite separate from the nipple was an irregular, lumpy, hard tumour, about half the size of an egg, and in the axilla was a large hard gland.

On May 2nd Mr. Smith removed the breast and the axillary gland; each was found to be occupied by a typical scirrhous growth.

Case 5.—L. S—, æt. 51, was admitted into St. Bartholomew's Hospital under the care of Mr. Smith on June 23rd, 1883. She was a healthy married woman who had borne and suckled nine children, and had never had whilst suckling, any trouble with the breasts. She said that three years before coming to the hospital the right nipple began to be chapped and sore, and that the

skin around it was red and irritable. This soreness continued in spite of treatment until the time of her admission, and four months before she came under notice she first discovered a lump in the breast.

An examination showed that the nipple had been almost entirely destroyed by ulceration, and that the skin around it was red, raw, and moist for a distance of about an inch. The affected skin was bright red and papillated, and was separated from the healthy skin by a definite sharp margin. Immediately beneath the nipple, and adherent to it, was a hard, irregular, lumpy tumour, rather larger than a walnut and tender to the touch. The axilla contained several enlarged and hard glands.

On June 20th the breast and the glands were removed, and were found on examination to be occupied by typical scirrhous growths, that in the breast involving the base of the nipple. The patient did well after operation.

Case 6.—E. K— was admitted into Lucus Ward, St. Bartholomew's Hospital, under the care of Mr. Savory on December 2nd, 1883. She was a healthy-looking woman, æt. 51.

For two years she had noticed the left nipple sore and "chapped," and for nine months had known of a tumour in the breast.

The nipple was found to be red, raw, and discharging; the areola was similarly red and raw in some parts, and covered with scales and crusts in others. Beneath the nipple was a tumour the size of an orange, very hard and adherent to the skin. In the axilla were several large, hard glands.

On December 22nd Mr. Savory removed the breast and the axillary glands, and found a mass of scirrhous cancer in each.

Case 7.—H. B—, æt. 44, was admitted into St. Bartholomew's Hospital under the care of Mr. Smith on June 1st, 1885. She said that she had first had trouble with her

gradually ast ten years previously whilst suckling, and that nipple abscess had then formed. From this trouble she had quite recovered, but five years later, whilst suckling the last child she had borne, the nipple again became cracked and sore, and since then had never got quite well. Two years before admission the soreness of the nipple became worse, and after that time a lump appeared in the breast. The patient had had seven children, and had otherwise been healthy. There was no family history of cancer.

On admission the place of the areola and a large extent of the skin around it were replaced by an irregular ulcer two inches and a half long and an inch and a half wide. The nipple had been destroyed, and the site of the nipple and the areola was occupied by a red, papillated, granular surface with sharply defined margins. In the breast beneath was a large, hard tumour, which was not adherent to the nipple or the ulcerated skin. There were several hard and enlarged glands in the axilla.

On June 3rd the breast and the glands were removed. The tumour was a typical scirrhous cancer, and the lymphatic glands were occupied by a similar growth.

Case 8.—S. A—, æt. 41, was admitted into St. Bartholomew's Hospital under the care of Mr. Cripps on March 8th, 1886. She was a healthy married woman, with seven children, and had never had any previous trouble with her breasts. She said that eighteen months before she came to the hospital she had suffered from soreness, which began around the left nipple and subsequently spread to the nipple itself. This got worse in spite of treatment, and after about a year's interval she first noticed a lump in the breast.

An examination showed that the left nipple had been to a great extent destroyed by ulceration. It was raw and ulcerated, and surrounded by a circle of granulation tissue discharging pus. Separated from the nipple and areola and situated in the inner half of the breast was an

irregular and ill-defined tumour as large as a walr lay in in the axilla were some slightly enlarged glands.

On March 16th the breast and glands were remov d A section of the former showed a typical scirrho carcinoma situated at a little distance from the nipple, but the glands did not contain any growth.

In June, 1888, the patient came back to the hospital with a small recurrence in the scar, but no sign of eczema or of enlarged glands. The growth was successfully removed.

Case 9.—P. C—, æt. 64, was admitted into President Ward in St. Bartholomew's Hospital under the care of Mr. Smith in December, 1887. She was an unmarried woman, and had suffered from soreness of the left nipple, without apparent cause, for six months. Two months later a lump appeared in the breast. Her family and personal history was good.

On admission the nipple was found depressed and raw, whilst the skin around it was red and weeping, and covered in patches with epithelial scales and crusts. There was no ulceration. Immediately beneath the nipple, and attached to it, was a hard tumour as large as a walnut, and with very irregular and knotty outline. The axilla contained one enlarged and hard gland.

On January 1st, 1888, the breast was amputated, and the enlarged gland was removed from the axilla. A section of the breast showed a typical scirrhous carcinoma involving the base of the nipple; the gland also was cancerous. The patient made a good recovery.

Case 10.—A. P—, æt. 57, was admitted into Lucas Ward under the care of Mr. Howard Marsh on June 13th, 1889. She said that two years and a half previously the right nipple began to be chapped and sore, and that this was accompanied by redness and a watery discharge from the skin of the areola. After this the nipple was

gradually eaten away by ulceration. Two years after the nipple began to be sore she noticed a lump in the breast. Her personal and family history was good, and she could not attribute the disease to any cause.

An examination showed that the right nipple had been completely destroyed by ulceration, and that the skin for an inch around had shared in the destructive process. The result was an irregularly circular ulcer with rather hard edges and base, and slight excoriation of the cuticle immediately around. To the right of the ulcer, and at a slightly higher level, was a tumour the size of a walnut, very hard, a little tender, and fixed to the superjacent skin. The axilla was free. On June 19th the whole breast was removed, and on section the growth was found to be a typical scirrhous carcinoma extending to the skin on the outer side of the breast, but not being continuous with the ulcer which occupied the position of the nipple. The patient made a satisfactory recovery.

Case 11.—H. C—, æt. 63, an unmarried woman, was admitted into St. Bartholomew's Hospital under the care of Mr. Morrant Baker on November 14th, 1890. She said that she had had a sore nipple for five years, and that within the last six months the areola also had become raw and sore. Two months before admission she first noticed a lump in the breast below the nipple. There was no family history of cancer or of injury.

She was a thin, healthy woman. The nipple was found to be partly destroyed by ulceration, and was much retracted. Its surface was red, raw, and granular. Around it the areola was red and congested, although not to any considerable extent. Here and there were small crusts, and in places the epithelium had been destroyed and a raw excoriated surface was exposed. There was a good deal of watery discharge, some burning pain, but no considerable irritation. The diseased area felt stiff and a little indurated on the surface. Below the nipple a tumour the size of a walnut could be

felt in the breast, and a hard and enlarged gland lay in the axilla.

The whole breast and the axillary glands were removed by Mr. Baker. The tumour was a typical scirrhous carcinoma, and the gland was similarly diseased.

Case 12.—An unmarried lady, æt. 60, came under the care of Dr. E. S. Tait and Mr. T. Smith in January, 1890.

She said that she had first noticed a soreness of the nipple five years previously, and that in spite of various treatment this had gradually got worse. Mr. Smith informed me that the nipple and areola presented the appearance typical of Paget's disease, but that no tumour could be discovered. He advised amputation of the breast, and this operation was performed on January 10th. After removal the breast tissue was found to be normal except at the orifices of the ducts, and no growth was found in any part.

Case 13.—The specimen in this case was sent to the museum at St. Bartholomew's with a statement that the eczema of the nipple had existed for some years, and that a growth in the breast had been noticed six months.

The tumour was found to be a scirrhous carcinoma, the areola was excoriated, and the nipple partly destroyed by ulceration. The growth in the breast was placed at some distance from the nipple.

Before venturing upon any remarks which the foregoing cases suggest it appears to be advisable to allude very briefly to the cases published by other writers in order to form some estimate of the available information.

I find that at the present time eleven cases have been recorded in England, two in Scotland, six in France, four in America, and one in Germany. With the exception of the last, clinical details are given as well as accounts of the morbid anatomy of the disease. It thus appears

that, although no records of cases occurred out of England for several years subsequent to Sir James Paget's paper on the subject, the disease is nevertheless widely distributed, and it is certain that it is much more common than these figures might lead us to think—a fact which is emphasized by the consideration that the cases I bring forward to-night are as numerous as all the other English cases hitherto recorded.

The whole literature of the subject has been so lately reviewed by Dr. L. Wickham[1] in an admirable monograph to which I shall again allude, that there does not appear to me to be any necessity for describing in detail cases already published. I am, however, indebted to Dr. Wickham's book for many references, and for a detailed account of the cases hitherto observed in France by himself and M. Darier.

From a study of the published cases the following deductions may be drawn. With regard to the *sex* of the patients, all the examples of the disease occurred in females, with one exception, that of a man aged 66, recorded by Forrest.

As to the age of the females *when they came under notice*, 3 were between thirty and forty, 5 between forty and fifty, 9 between fifty and sixty, 4 between sixty and seventy, 1 between seventy and eighty. Total 22.

The youngest patient was aged thirty-three, and the oldest seventy-two, and in the former the disease of the nipple commenced at the age of twenty-eight.

In nine cases there was no tumour in the breast. In the remaining fourteen the mamma was the seat of cancerous growth.

The duration of the disease of the nipple in the nine cases where there was no tumour was in one six months, in one two and a half years, in one three years, in one five years, in one nine years, in one ten years, in one eleven years, in one twenty years, in one a doubtful period.

In the fourteen cases where the disease of the nipple

[1] 'Maladie de la peau, dite maladie de Paget,' par le Dr. Louis Wickham.

was complicated by a tumour in the breast, the duration of the disease of the nipple *before operation* was in one case fifteen days, in two cases six months (one a male), in two cases one year, in one case one and a half years, in three cases three years, in one case four years, in three cases six years, in one case twelve years.

In eight cases there was *ulceration* and destruction of a part or the whole of the nipple as well as of the skin of the areola, and of that in its neighbourhood to a varying extent.

A similar analysis of the cases recorded by myself shows the following results.

Age when seen by surgeon, 1 between thirty and forty, 3 between forty and fifty, 5 between fifty and sixty, 3 between sixty and seventy. The youngest patient was aged thirty-six and the oldest sixty-four. The earliest age at which the disease *commenced* was thirty-five.

In two cases there was no tumour in the breast. In the remaining eleven the mamma was the seat of cancerous growth.

The duration of the disease of the nipple in the two cases where there was no tumour was in one three years and in the other five years.

In the eleven cases where the disease of the nipple was complicated by a tumour in the breast, the duration of the disease of the nipple *before operation* was in one case six months, in one case one year, in one case one year and a half, in one case two years, in one case two years and a half, in three cases three years, in two cases five years, in one case seven years.

In seven cases there was no ulceration, whilst in the six others more or less of the nipple and of the areola had been destroyed.

It is further to be noticed that in none of the cases recorded by myself or by other writers (with one exception) was the soreness of the nipple the result of lactation or its attendant troubles. In most cases the mamma had not been used for suckling for many years, whilst several of

the patients were single women who had never been pregnant. There was seldom any history of injury or of cancerous tumours in other members of the family.

With regard to the appearance of the diseased nipple and areola, it is clear that the lesions are not those of simple eczema. It is true that in the earliest stages of the disease there may be appearances which cannot be certainly differentiated from those of eczema, but as the process extends certain definite distinctions can be established.

At first there is a branny desquamation, with slight watery discharge which may be noticed either on the nipple itself or else on the areola. After a variable time small scales and crusts form, and when these are cast off a raw and excoriated surface may be exposed, which may again be covered by a scab. As the disease spreads it does so in a definitely eccentric fashion, so that the nipple remains throughout the centre of the affected area. In all recorded cases there has been no formation of vesicles or pustules, and the margin of the diseased tissues is sharply defined. Although commencing in, and at first confined to the nipple and areola, the morbid process may extend to and involve a considerable area of the skin covering the breast. In all cases there is slight stiffness and induration of the affected parts, so that the skin cannot easily be pinched up or thrown into folds.

In the later stages of the disease the nipple becomes retracted, and may be withdrawn to such an extent that its summit becomes depressed below the level of the surrounding integument. The surface is not equally denuded of epithelium, and small particles of the latter may remain as islands in the area of excoriation.

In most of the advanced and chronic cases the surface of the nipple becomes ulcerated, and this ulceration extends to the areola, and thence to the skin around it. As a result of this the whole nipple may be destroyed, and a large circular or oval ulcer with a definitely papillated surface may occupy the site of the areola. From

the commencement of the disease the affected parts are much more red and congested than are eczematous tissues, and the redness becomes more marked in the later stages of the affection. Most patients complain much of continued burning pain, but not of severe itching. In many cases the disease, after being comparatively quiescent for a long time, spreads with greatly increased rapidity. This is especially the case before the development of a cancerous tumour in the breast.

In all the cases in which a cancerous tumour of the breast has succeeded to the disease of the nipple and areola, the new growth has run the usual course of a scirrhous carcinoma, and has been found on clinical examination to present the usual characters of this form of tumour.

Histology.—The microscopical anatomy of Paget's disease was first described by Mr. Butlin,[1] and his descriptions have been confirmed by most other investigators. The lesions are essentially inflammatory in their nature, and differ in degree and extent according to the duration of the morbid process.

Mr. Butlin sums up his observations with the following conclusions as to the conditions found :

(*a*) Proliferation with thickening of epithelium.

(*b*) Infiltration of subcutaneous tissues.

(*c*) Filling up of ducts, and infiltration of leucocytes around.

(*d*) Acini enlarged and filled with epithelium.

Dr. Thin says,[2] "It is in this weaker layer of the connective tissue of the cutis (pars papillaris) that the changes take place which produce the disease of the nipple and areola which I am discussing. These changes consist in a complete destruction of the connective tissue of the layer, the space which is occupied being filled with a dense mass of exudation cells. The persistence of the epidermis in a more or less complete state distinguishes

[1] 'Med.-Chir. Trans.,' vols. lix and lx.

[2] 'Brit. Med. Journ.,' 1881, vol. i, pp. 760 and 798.

the condition from that of ulceration. . . . The affection of the nipple is neither eczema nor any known specific skin disease, but a destructive dermatitis of the papillary layer."

Messrs. Duhring and Wild[1] describe similar changes, and lay stress on the fact that the process appears to be most intense in the papillary layer of the skin, whence it spreads into the breast tissue and towards the surface.

Various other authors have described similar appearances, and Butlin and Thin have further figured and described certain peculiar appearances in the surface epithelium which they believed were due to endogenous cell formation.

The conclusions of these observers as to the inflammatory nature of the process have lately been confirmed by Darier and Wickham, but the observations of these authors which are of the most interest at present have reference to the presence of parasites in the affected parts. The parasites in question were first described by Mr. Darier, and in six other cases have been demonstrated by Mr. Wickham. These gentlemen are of the opinion that the vacuolated cells described by Butlin and Thin are really psorosperms, and have figured these parasites in the epithelial cells of the diseased tissues.

Mr. Wickham says that the parasites are always enclosed in epithelial cells, and present under various forms. In many cells there are only irregular protoplasmic nodules, which cannot be certainly proved to be psorosperms.

In a further stage of development the psorosperm distends the cell in which it is placed, and thrusts the nucleus to one side. It then presents itself as an oval or round body with a definite envelope, sometimes containing several nuclei. In many specimens the protoplasm of the parasite is shrunken and retracted, so that it does not fill its capsule, and leaves a clear space or vacuole. The contracted protoplasm usually occupies the centre of the capsule ; in other cells the protoplasm is collected to

[1] 'Amer. Journ. of Med. Sci.,' July, 1884.

one side of the capsule. At a later stage of development it appears that the capsule bursts and discharges its contents. The capsule in its typical form has a double contour.

With regard to the condition of the breast tissue and of the cancerous growth, all observers, with the exception of Dr. Thin, describe the tumours found as "scirrhous cancers" or "spheroidal-celled carcinomas." Dr. Thin considers that the specimens examined by him were examples of duct cancer.

Mr. Wickham describes in two of his cases ingrowth of the interpapillary processes, and the presence of epithelial nests in some sections, and considers that in these specimens there was a commencing epithelioma. Inasmuch, however, as these cases did not clinically present the usual appearances of epithelioma, and did not in any way run the course of this disease, I do not think the evidence adduced justifies the conclusion arrived at. I am all the more inclined to this opinion because it is well known to all pathologists that at the edge of any old ulcer the interpapillary processes are large and branching, and are frequently mistaken by inexperienced observers for the typical ingrowth of an epithelioma.

The changes seen in breasts which have not become cancerous may be briefly summed up as inflammatory in their nature. They consist in—

(*a*) Proliferation of the epithelium of the ducts and acini, the cells of which may fill the tubes.

(*b*) Cell infiltration of the connective tissue, and subsequent formation of fibrous tissue, first around the ducts, and subsequently deeper in the breast.

With regard to the cases I have myself recorded, I would say that, from an examination of a very large number of sections, I am of the opinion there is little to add to the generally accepted descriptions of the affected tissues.

I have found in the earliest cases an infiltration of the papillary layer of the skin with leucocytes, and in many

specimens I have found the blood-vessels most unusually large and numerous. In such specimens the surface epithelium is in places thickened and desquamating, whilst the interpapillary processes are large, and penetrate deeper than is usual into the skin. The sebaceous and sweat glands do not show any change.

At a later period the epidermis becomes gradually thinned away, in proportion as the derma beneath is destroyed by the small cell-growth, and in the final stages of the process the whole epidermal layer is destroyed, and a granulating surface is exposed. In some specimens there is a definite papillary growth, caused apparently by a swelling and increase in size of the papillæ of the derma, which are covered at first by thickened epithelium.

As to the presence of psorosperms, I would first of all premise that in all my cases except the last one my original sections had not been specially stained or prepared with a view to the discovery of these parasites, the presence of which has only been described very recently. After reading Mr. Wickham's work, however, I prepared a very large number of sections taken from the breast removed by Mr. Baker in Case 11. I had also fortunately kept portions of tissue from six others of my cases, and these were again cut and re-examined. I shall allude later to the methods of preparation employed, but must take the present opportunity of expressing my indebtedness to Mr. Miller, one of my clerks, for the great skill and care exhibited by him in the preparation and mounting of many of the sections.

Considering first of all the specimens obtained from Case 11, I was at once struck by their similarity to the drawings made by Mr. Wickham. Indeed, so close was the resemblance that some of his drawings might well have been made from sections taken from this case. It was evident on even the most superficial observation that the bodies he had described as parasites were to be found in very large numbers in these specimens.

Under a half-inch objective the epidermis was seen to

contain innumerable oval or rounded bodies twice or three times as large as an epithelial cell. Many other smaller, but evidently similar, bodies could be seen in neighbouring cells, and it was noticeable that almost all the oval-shaped bodies were arranged with their long axes at right angles to the free surface. These bodies were confined to the epidermis, and were nowhere seen in the cutis vera. They were more numerous and well marked in the deeper parts of the epidermis and in the interpapillary processes than in the more horny layers, and some of them, though very few, were to be seen at the orifices of the milk-ducts, though not in the deeper parts of these tubes.

The bodies in question consisted apparently of a well-defined wall bounding a central clear cavity or cyst, in which lay protoplasmic granules and nuclei. In some of the cysts there were several nuclei which almost or quite filled the cavity, but in the majority the protoplasmic contents were scanty, and either occupied a small area in the centre or were collected to one side of the space.

Under a higher magnifying power it was seen that many of the smaller of the bodies mentioned could not be certainly distinguished from the surrounding protoplasm of the cells within which they lay. The larger bodies looked at first more like vacuoles in the epithelial cells than like psorosperms, yet it was evident that they were not merely vacuoles and nothing else. The regularity of their distribution in the deeper epithelial cells, the presence of several granular nuclei inside them, their presence in the areola in almost all the cases examined, and their absence from the surrounding skin convinced me that they could not be accounted for merely by a process of vacuolation.

On the other hand, after examining a series of psorosperms from the livers of rabbits it was evident that these bodies were not typical psorosperms, for in hardly any of them could I distinguish the envelope with a double contour which characterises these parasites, and upon which much stress is laid by Wickham.

It appears to me, after a very careful examination of some hundred sections, that it is impossible to affirm with absolute certainty that the bodies described by Wickham and myself are clearly psorosperms, and I think that the matter is one on which there may well be legitimate differences of opinion. My own opinion is that these bodies are probably psorosperms, and I am naturally influenced in arriving at this conclusion by the fact that in no other pathological process affecting an epithelial covered surface have I ever seen anything which in any way resembles the appearance presented by the innumerable bodies I have described as lying in swarms in the epithelial cells. It is certain that such bodies as these are not to be found in cases of simple eczema, and they are certainly not present in cases of real eczema of the nipple, as I have had an opportunity of proving by the examination of a breast which was removed for eczema and suppuration following on lactation. The appearances presented in a typical case of Paget's disease are in fact so striking that, when once familiar with the appearance under the microscope, no observer could fail to recognise other specimens of the disease, and could not mistake the sections for those of simple eczema or ulceration.

In no case have the psorosperms in a case of Paget's disease been successfully transplanted into animals, but it should also be remembered that experiments with undoubted psorosperms from rabbits have similarly failed.

With regard to the proportion of cases in which the psorosperms were found, I may say that in all cases where many sections were made the appearances I have described were to be seen, although some sections showed no evidence of parasitic growth, whilst others taken from the same breast showed many; the total number of barren sections was, however, very few. In the seven breasts, in each of which I had but four or five sections preserved, and of which I had no more material for further examination, I found the parasites in greater or less numbers, although in two cases they were so few as to require a

long search to establish their presence with certainty. In these cases also it should be remembered that no special staining methods had been employed.

There is but little to describe further as to the microscopical appearance of the breast tissue itself in these cases. I have in most found evidence of proliferation of the duct epithelium, although in some cases it was but slight, and in most breasts where there has been a tumour the remainder of the mammary secreting tissue has been normal. In those cases where the tumour has been developed in a part of the breast far removed from the nipple, I have generally found no change in the intermediate mammary tissue, and such changes as have been noticed have been limited to catarrh of the ducts. In many specimens I have found much small-cell infiltration around the inflamed ducts, and in some cases this cell-growth was replaced by fibrous tissue.

In all the cases I have examined the tumour was a typical spheroidal-celled alveolar carcinoma, evidently originating in the epithelium of the acini, and when the lymphatic glands were involved they contained similar growth. In none of the many sections examined did I find any evidence of psorosperms in the cancerous growth either in the breast or in the lymphatic glands, although careful search was made in order to confirm this observation.

In staining my sections I used hæmatoxylin, osmic acid, methylen blue and rubin, eosin and picro-carmine.

The true pathology of "Paget's disease of the nipple" is a matter of considerable importance if the disease is to be treated rationally, and if the cancerous growth which so often ensues is to be obviated.

Sir James Paget, Mr. Butlin, Dr. Goodhart, and others of the earliest observers did not make any suggestions as to the cause of the disease of the nipple, and it was left to Dr. Thin to be the first to formulate a theory as to its causation. This observer suggested that the disease of the nipple was not itself primary, but that it was the

result of a cancerous growth originating in the ducts, and was caused by the irritation of the discharge set up by the tumour; or, as Dr. Thin puts it, "the hypothesis is that a change takes place in the columnar epithelium at or near the mouths of the ducts, which exerts a destructive influence on the adjacent connective tissue, and is in reality the first stage of cancer." As already stated, Dr. Thin considered that the tumours in the breast were "duct cancers."

It is now many years since Dr. Thin first propounded these views, and I do not know whether he still holds the same opinions. I think, nevertheless, it as as well to point out that the observations published since his papers do not lend any support to his views, and I would submit the following arguments against their acceptance.

1st. There is no evidence in any recorded case that such a discharge from the nipple as is supposed to occur has ever been seen; and although it was carefully inquired for in all my cases, in none of them was any discharge present.

2nd. Duct cancers are soft, vascular, and friable growths, whilst in all the recorded cases of Paget's disease, as well as in those I have seen, the tumours have been hard and scirrhous. Duct cancers are much less liable to affect the glands and to disseminate than were the tumours described by other authors or seen by myself.

3rd. In a recent volume of 'St. Bartholomew's Hospital Reports'[1] I have described a series of six cases of "duct cancer," and have collected the reports of other published cases. In almost all of them there was discharge of much blood-stained fluid from the nipple, but in not one of them was there any "Paget's disease."

4th. The histories of undoubted cases of duct cancer are very different from those of Paget's disease, and it is not possible to believe that in cases where this condition of the nipple has lasted for as long as nine, ten, or even twenty years without the development of any tumour,

[1] Vol. xxvi, p. 263.

there has been nevertheless a cancerous growth during the whole of such a period.

5th. In cases where the mamma has been removed before the development of any evident tumour (with the exception of one case of Dr. Thin's), no cancer has been found on subsequent examination of the breast, and such patients have remained subsequently free from tumour growth. On the other hand, when definite tumours have been present the course of the case has been that which is common in other examples of scirrhous carcinoma.

It is then evident that some other origin must be sought for a disease which is so evidently *sui generis*, and I have already pointed out that the cause is not to be found in the inflammatory lesions of lactation, nor in any antecedent condition of the breasts such as chronic mastitis.

Accepting, therefore, that the morbid condition of the nipple precedes any morbid change in the mamma, it is evident that the cause of a lesion so persistent and chronic must itself be of the same nature. It is certain that no transient cause could account for so long lasting a disease. Further, but very little consideration is required to negative the suggestion that the cause is a constitutional one, for the patients whose breasts are affected are otherwise healthy, there is in almost all no family history of cancer, and the fact that the area of skin involved is so small a part of the whole body surface, added to the equally important fact that but one nipple is commonly attacked, indicates that the origin of the affection is not to be sought in any constitutional predisposition.

Quite lately, however, Drs. Darier and Wickham have attributed the disease to the presence of the psorosperms in the diseased skin already alluded to, and their observations have been confirmed by Mr. Jonathan Hutchinson, jun., in a case recorded by him in the last volume of the 'Transactions of the Pathological Society.' I have already expressed my opinion on the presence of psorosperms in my own sections.

With regard to the parasites themselves, it may be said

that psorosperms are found under very different forms, and for full details of their mode of growth and their varieties I would refer to the admirable paper by Dr. Delépine published in vol. xli of the 'Transactions of the Pathological Society.' From the observations of this author, who studied the parasites in the livers of rabbits, it would appear that psorosperms may be found—

1st. "As small, round, oval or elongated, structureless masses, mixed with a number of granules, which are of smaller size, and possibly indifferent." These bodies, when inside epithelial cells, cannot be differentiated from protoplasmic granules.

2nd. "After a short time, however, bodies looking very much like nuclei appear in various parts of the [epithelial] cells. The cells containing these bodies are larger than the normal ones. These nuclear-looking bodies are probably nothing more than small protoplasmic masses contained in a large vacuole-like cavity; when the small mass is slightly contracted a space remains between it and the walls of the cavity, and this space looks like a membrane with double contour."

3rd. "The body now assumes distinctive features, which render its recognition easy. The small amœboid mass contains a nucleus-like body, which itself encloses one large nucleolus or several rounded granules. The protoplasma surrounding the nucleus is coarsely granular, and many of the granules stain black with osmic acid, and brown with iodine."

4th. "The parasite assumes a distinct ellipsoidal shape, and becomes larger than an ordinary epithelial cell, and distends the enlarged cell in which it is lodged. An external layer (ectosarc) is at this stage distinct from the granular endosarc; the granules are large, and distributed equally all over the body of the organism: the nucleus is indistinct, and seems to be replaced by a large mass of highly refractive granules, which stain quite black with osmic acid and dark brown with iodine. A nucleus may, however, be demonstrated at this stage by staining the

specimen with rubin and double staining with methylen blue. . . . The organism is distinctly contractile, and may assume a nearly globular shape, or an elongated, almost vermiform appearance. It is often irregular, being, for instance, flat on one side and convex on the other. The mass may also contract within the cavity containing it, so as to leave a space between itself and the protoplasma of the cell. . . . They measure from 30 to 35 mm. in length by 15 to 20 mm. in width."

The parasite may escape from the cell containing it, or its capsule may burst and allow of the escape of the protoplasmic contents.

Psorosperms are widely distributed in the animal kingdom, and are found in amphibians, fishes, birds, and mammals. In fish they are found in all the organs ; in the higher Vertebrata chiefly in the alimentary canal and the liver. In rabbits they are especially common in the latter organ, and according to Dr. Delépine occur in 92 per cent. of all animals examined.

The next point for consideration is the part played by the parasite in causing the diseased conditions amongst which they are found ; for, assuming that they really occur in all cases of Paget's disease, it by no means follows that they cause all the lesions associated therewith.

Three theories may be formulated.

1st. The parasites may be implanted in the diseased skin, and may grow on a surface the cells of which are already damaged.

2nd. The parasites may cause the disease of the nipple.

3rd. The parasites may be the direct cause of the cancerous tumours.

With regard to the first suggestion I would say at once that I do not consider the presence of the parasites to be merely fortuitous. Considering that they have been found in all the cases examined in France, in most of the specimens prepared by myself, and that earlier writers have described appearances which might well be due to the presence of psorosperms, I am myself of the opinion

that these facts alone are sufficient to negative the theory that their presence is merely a matter of chance. It is further certain that psorosperms are not to be found in ulcerated or raw surfaces as a rule, and I have myself failed to find them in any other tissues or diseased organs I have examined.

With regard to the causation of cancer by psorosperms Dr. Wickham appears to me to hold opinions which are not in any way supported by facts. He says that he considers the parasites to be the *direct* and specific cause of the cancer, and in support of his contention states that he has found the parasites in the cells of the new growth. If, however, Dr. Wickham's work be carefully examined, it will be seen that he merely depicts the presence of the psorosperms in the cells of what he describes as "epithelioma," and not in the cells of any definite tumour found in the breast. As I have already stated that there is no good reason for believing that epithelioma really existed in his cases, it is nearly certain that the cells in which the parasites were seen belonged to interpapillary processes.

I am the more strongly of the opinion that the psorosperms have no specific influence on the causation of the cancer for the additional reasons that out of many hundred sections examined none of these parasites were found in any part of a scirrhous tumour ensuing upon Paget's disease, nor did I find any traces of them in the secondary growths in the lymphatic glands.

As additional arguments against the theory that the psorosperms are the direct and specific cause of the cancer, it may fairly be urged—

1st. That, in numerous cases where the disease of the nipple was of long standing, they have probably existed for many years in the nipple and the areola, and have yet caused no cancer, whereas if they possess some definite specific influence this could scarcely be the case.

2nd. That in the many animals in which they exist they are not associated with tumour growth.

3rd. That in the vast majority of cases of cancer in

the human subject psorosperms are certainly not present.

4th. That when they have been found in the human subject, apart from cases of Paget's disease, *e. g.* in the liver, the ureter, &c., they have not been associated with cancer.

I have, therefore, no hesitation in concluding that the problem of the etiology of cancer has not been solved by the discovery of these parasites in the nipple, and that they have no specific influence in causing cancerous growth.

The facts, as far as we know them at present, rather seem to me to point to the conclusion that the disease of the nipple is the direct result of the presence of psorosperms, and that the raw and irritated surface bears the same relation to the development of cancer subsequently as does an old ulcer of the leg or of the tongue to an epithelioma.

A further problem to be solved is how the parasites obtain a footing in the areola, and why they should attack this portion of epidermis. To this, I think, we have at present no satisfactory answer. It is, of course, reasonable to suggest that the skin of the areola probably shares with the mammary tissue in the degenerative changes which occur late in life, and thus becomes predisposed to morbid processes, and less able to resist parasitic growth.

But if this theory be accepted it must still be explained why more evidently damaged skin in other parts of the body is not attacked in a similar way, for it is certain that portions of eczematous or ulcerated skin are not commonly attacked by psorosperms. The mode of infection of the breast is again a matter in which as yet no evidence is forthcoming, and no good reason has yet been assigned for the depth beneath the areola at which the cancerous growth is sometimes placed.

The development of a tumour at some distance from the irritation which is supposed to cause it is not paralleled by the known histories of growths in other parts of the body.

DESCRIPTION OF PLATE VI.

Thirteen Cases of Paget's Disease of the Nipple (Anthony A. Bowlby).

Fig. 1.—Section of the skin of the areola from a case of Paget's disease. The section is taken from a part beyond the area of excoriation, and shows in the midst of the normal epithelial cells numerous oval or rounded spaces with well-defined margins. Some of these spaces are occupied by nuclei, others are empty ($\frac{1}{2}$ inch Crouch).

Fig. 2.—Section through the same area of skin under a higher power ($\frac{1}{6}$ inch Crouch). It shows one of the rounded spaces occupied in part by a granular mass of protoplasm with a nucleus. The space has been formed by the distension of an epithelial cell, and the nucleus of the cell can be seen at the lower part of the space and a little to the left of it.

Fig. 3.—Section showing a cavity occupied by three oval granular masses surrounded by protoplasm (Crouch $\frac{1}{6}$ inch).

Fig. 4.—Section of an empty vacuolated cell, greatly flattened, the nucleus of which may be seen to the left of the space.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 147.)

Fig 1

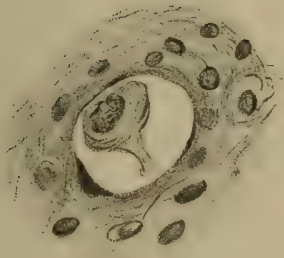

Fig 2

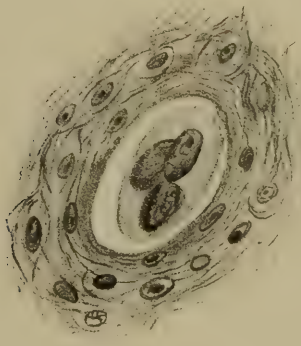

Fig 3

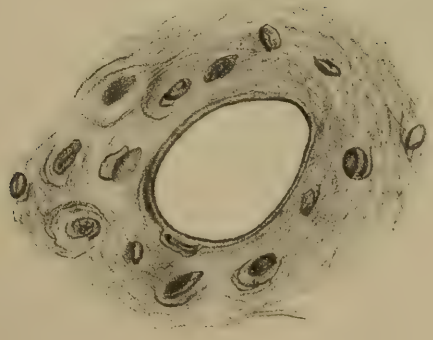

Fig 4.

ON THE CHANGES

OBSERVED IN

HEALTHY MEDIUM-SIZED ARTERIES AND IN TENDON LIGATURES

DURING THE FIRST FOUR WEEKS

AFTER LIGATION.

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I. Preliminary Remarks.

Ten years ago, one of us brought before the Society a paper[1] in which the behaviour of a tendon ligature was dealt with. In the course of the discussion that followed, a promise was made that the results of further observations on the same subject should also be communicated, in due course, to the Society. The long delay in the fulfilment of this promise has been due to various causes. As a matter of fact, indeed, all the results of the investigation which we now bring forward were already obtained early in 1886. The reading of Messrs. Ballance and Edmunds's paper[2] before the Society was one reason that deterred us from launching our paper during the same session. With the exception of a few notes published by one of us concerning the organisation of the clot,[3] we allowed the matter to drop for the time, being of opinion that the subject had perhaps been sufficiently under discussion,

[1] Dent, 'Med.-Chir. Trans.,' vol. lxiv, 1881, p. 231.
[2] 'Med.-Chir. Trans.,' vol. lxix, 1886, p. 443.
[3] Delépine, 'Brit. Med. Journal,' 1887.

and fearing that it might prove wearisome. We are, however, so convinced that some of our facts have a distinct bearing upon the practice of ligation of arteries in their continuity, that it does not seem right to withhold them any longer.

Since the immediate changes occurring after ligation have been studied and described by numerous observers, *e. g.* Stilling, Billroth, Cornil, Ranvier, and Shakespeare, as well as by many other old and recent writers, it was not thought necessary to re-investigate this branch of the subject. Moreover the main object of the investigation was not the study of the causes of thrombosis.

Our inquiry was directed chiefly to ascertaining the effects of a certain form of ligature applied tightly to the vessel. Earlier than the fifth day after ligation, little of practical utility could be added to the knowledge already gained. Further, if the method was a good one, all the changes necessary to produce complete and permanent obstruction were expected to be completed within three weeks. These considerations will explain the limits of time within which our observations extend.

II. Experiments.

Nature of the Experiments.

The femoral arteries of six sheep were tied with chromo-carbolised kangaroo tendon by Professor E. C. Stirling, in Australia, on September 7th, 1884.

The 1st sheep was killed 5 days after the operation.

2nd	,,	9	,,	,,
3rd	,,	13	,,	,,
4th	,,	17	,,	,,
5th	,,	20	,,	,,
6th	,,	23	,,	,,

In every case the ligature was tightly tied, so as to cause rupture of the internal coats. The operation, per-

formed with antiseptic precautions, was successful in all cases; all the wounds healed without suppuration. After removal from the body the ligatured portion of the arteries with part of the surrounding tissues was placed in $\frac{1}{6}$ per cent. chromic acid mixture, then in methylated spirit in the usual way, and after this the specimens were sent to England.[1]

Preparation of the Specimens examined.

In order to display the relations of their parts, all the specimens were carefully bisected in such a way as to include in the plan of section the constricted part of the artery, the knot of the ligature, and the traumatic region (that is the line of the incision made for the purpose of exposing the vessel). It may be remarked here that this section was tolerably easy to carry out in the specimens obtained soon after the operation. In the last three, however, the difficulty was very great, owing to the deformity caused by contraction or other causes, which had produced more or less marked tortuosity of the vessel. One half of the artery was kept for the purpose of cutting longitudinal sections for microscopical examination. The other half was used for the purpose of cutting transverse sections at various levels.

It was found afterwards that by far the most useful sections were the longitudinal ones, especially those passing through the whole course of the constricted portion of the artery, showing the knot of the ligature on one side. In addition to these two modes of preparation, small portions of the ends of the ligatures were teased separately in a few cases.

[1] The effects of age, sex, state of the individual, or other influence of the species of animal or of disease do not form part of this investigation; any reference to such questions has been derived from other sources.

III. Macroscopical Appearances of the Arteries after Ligation.[1]

1. *Contents.*

In all cases the proximal side of the artery is distended with coagulated blood, modified by changes which will be described further on. In one instance the distal side of the artery also contains a small decolorised clot similar to that found on the proximal side. Up to the seventeenth day the proximal side of the vessel is larger in diameter than the distal side, which is much collapsed and empty (which means evidently that this part of the vessel contained fluid blood during life); after the seventeenth day the two segments are about equal, and gradually become more and more contracted.

In the fifth day's specimen (Fig. 1) the clot is dark, and has a convex oblique surface, extending nearly as far up as the first collateral branch above the seat of ligature. This main clot, which fills up the whole lumen of the artery, is capped by a small elongated finger-like process of decolorised clot, which is itself separated from the walls of the artery by a coarsely fibrillated, loose, mottled coagulum. This small digital clot is not axial, but occupies the half of the lumen of the vessel which is opposite to the opening of the collateral branch. It also extends for a short distance above the level of this opening. It is evident from these appearances that the thrombus was not formed all at once.

In the ninth day's specimen (Fig. 2) the clot occupying the proximal portion is smaller than in the previous specimen, but still large. Its surface is convex, but not so much as on the fifth day. Its colour is still dark, there being

[1] We have described the macroscopical appearances of our specimens separately in order to render the comparison of our results with those of older writers more easy.

only a thin layer of laminated decolorised coagulum on its surface. The small additional clots described in connection with the proximal end of the last specimen are

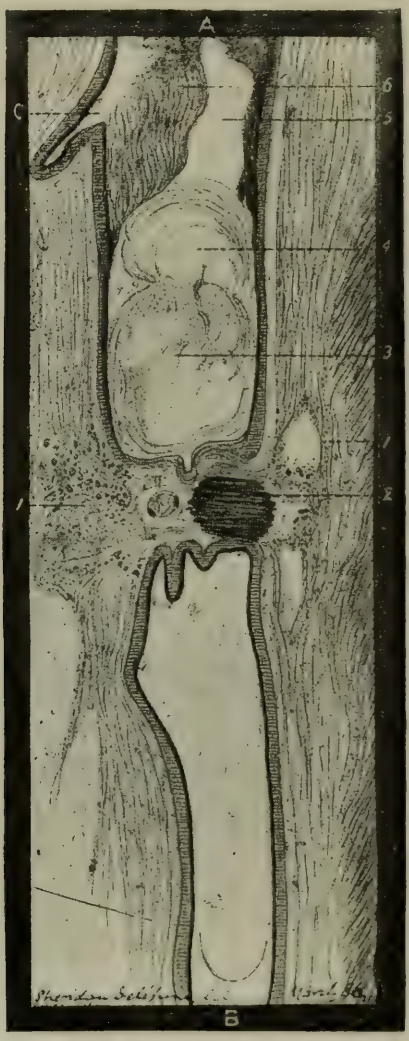

FIG. 1.—A. Proximal end of the artery. B. Distal end of the artery. C. First collateral branch above ligature. 1. Traumatic region. 2. Ligature. 3. Distal or oldest part of the clot. 4. Intermediate portion of the clot. 5. Proximal or digital projection of the clot (recent part). 6. Imperfectly coagulated blood; loose mottled clot. × 5.

not found in this case. Irregular elongated fusiform spaces, occupied by dark recently coagulated blood, are found between the sides of the main clot and the walls of the vessel.

In the thirteenth day's specimen (Fig. 3) the proximal portion contains a very small decolorised plug, the surface of which is distinctly concave, with a slight elevation at the bottom of the concavity.

FIG. 2.—A. Proximal part of the artery. B. Distal part of the artery. 1. Traumatic region. 2. Ligature. 3. Distal part of the clot. 4. Intermediate part of the clot. 5. Proximal part of the clot. × 5.

On the seventeenth day (Fig. 4) the plug is very small; its surface is concave; it is almost completely decolorised, having only a pale brownish colour.

NOTE.—The drawings (Figs. 2 and 3) representing the macroscopical appearances of the vessels were taken immediately after the first longitudinal section had been carried through. This section was purposely made on one side of the middle line, so that the constricted portion of the vessel cannot be seen. This was reserved for the microscopical sections (*vide infra*).

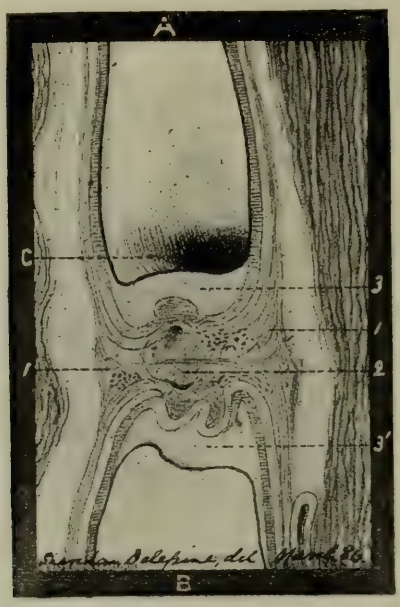

FIG. 3.—A. Proximal part of the artery. B. Distal part of the artery. C. First collateral branch above the ligature. 1. Traumatic region. 2. Ligature seen by transparency. 3. Proximal plug. 3′. Distal plug. × 5.

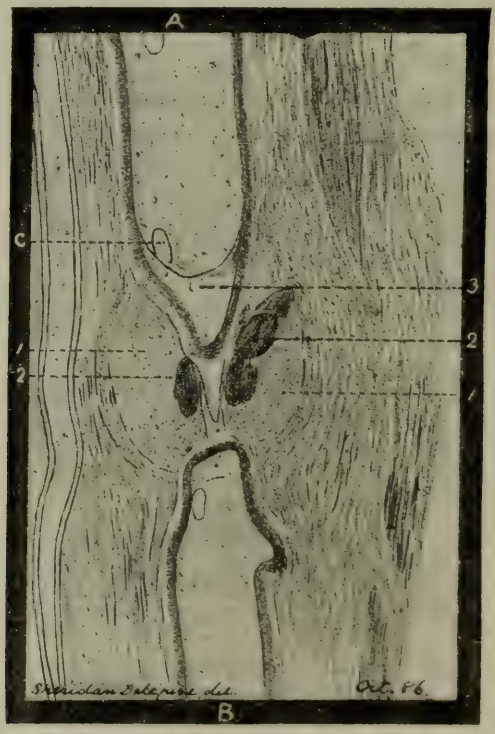

FIG. 4.—A. Proximal part of the artery. B. Distal part of the artery. C. First collateral branch above the ligature. 1. Traumatic region. 2. Ligature. 3. Plug. × 5.

In the twentieth day's specimen (Fig. 5) the plug is small, decolorised, but not concave, being almost perfectly flat.

In the twenty-third day's specimen (Fig. 6) the plug is a little larger than in the last case, whitish, fibrous-looking, markedly concave—in fact, infundibuliform.

2. *Walls of the Vessel.*

(1) The *intima* can be almost recognised with the naked eye, and very easily by means of an ordinary pocket lens,

FIG. 5.—A. Proximal part of the artery. B. Distal part of the artery. 1. Traumatic region. 2. Ligature. 3. Plug. × 5.

as a dark (owing probably to its being transparent) thin band, limiting the lumen of the artery both on the proximal and distal portions. In the neighbourhood of the ligature it is puckered, and its divided end curls inward, forming a mammillated projection into the lumen of the artery. This is the case both on the proximal and distal side of the constriction.

(2) The *muscularis* is also distinct in every case. It looks darker than the adventitia, and seems to be torn as

well as the intima by the ligature, being apparently almost entirely absent from the constricted region of all the arteries.

(3) The *adventitia* is distinct in all cases, is folded in the neighbourhood of the ligature, and is the only coat which remains constantly at the seat of ligature. In no case does it show any evidence of a tendency to give way.

FIG. 6.—A. Proximal part of the artery. B. Distal part of the artery. C. First collateral branch above the ligature. 1. Traumatic region. 2. Ligature. 3. Plug. × 5.

3. *Perivascular Tissue.*

The tissues adjacent to the artery and ligature are involved over a remarkably small area.

In the fifth day's specimen the affected area could easily be covered by a circle having for radius the diameter of the artery (coats included), and for centre the middle

part of the constricted portion of the artery (or rather of the side of it in contact with the knot of the ligature).

A circle having for radius twice the diameter of the artery, and the centre of which would be in the midst of the knot of the ligature, would just cover the whole of the affected parts in a section passing through the middle of the vessel *nine days after ligature.*

After the seventeenth day this area becomes rapidly smaller, the changes giving rise to what seems to the naked eye simply a fibrous thickening of the perivascular sheath.

The changes noticeable to the naked eye are not very striking; they are chiefly due to displacements produced by the incision, the dragging or drawing in of the tissues by the tightened ligature, the projection of the knot on one side of the artery, the accumulation of new material around the ligature (most noticeable in the ninth day's specimen), and finally a certain amount of discoloration and alteration of structural appearances, due at first to extravasation and exudation, and finally to cicatrisation and vascularisation.

4. *Ligature.*

The ligature is very distinct in all specimens, but is more conspicuous in those removed late after ligation than in those removed a few days only after operation. This is due to the fact that after being embedded in the tissues for some time the prepared tendon assumes a dark brown colour. On the other hand, in recent specimens the tissues surrounding the ligature have a darker colour than at a later period.

In the ninth day's specimen the ligature, where it does not form a large mass as in the region of the knot, is almost of the same colour as the surrounding tissues. In this case the parts of the knot do not hold together as well as in any of the others. The tendon has a tendency to split into fine fibrils, so that it is almost impossible to

obtain a good section of it. The surrounding products hold so badly together that the ligature seems to be quite loose in the midst of a small cavity. In the other specimens it is, on the contrary, tightly held by the inflammatory products, and is distinctly encapsulated after the thirteenth day.

In connection with the *ligature knot* an interesting appearance is produced in several of the vessels; where the knot presses upon the arterial walls these bulge outward very much less than on the opposite side, where the walls are not so supported. There is, however, no distinct evidence that this absence of support leads to any undue distension of the coats.

The folding of the arterial walls is also more marked on the superficial side of the vessel (that is, the side which is in contact with the knot). This causes a certain obliquity of the plan of section to certain parts of the walls, and leads to an apparent difference of thickness of the walls on both sides, an appearance which must not be attributed to any giving way of the walls on the thin side.

It can therefore be said generally, from a naked-eye examination of the specimens, that in all cases—

(1) The occlusion of the artery was perfect.

(2) The ligature, except in the second case, gave rise to very little irritation.

(3) The ligature showed no tendency to be absorbed.

(4) The walls of the artery did not show any tendency to give way.

(5) The clot as such had practically disappeared after the thirteenth day, and had been replaced by a fibrous-looking plug.

IV. Microscopical Examination of Specimens.

Fifth Day's Specimen (see Figs. 1, 7, 9, 10).

General remarks.—The occlusion is perfect. The ligature can be distinctly recognised; it is in perfect contact

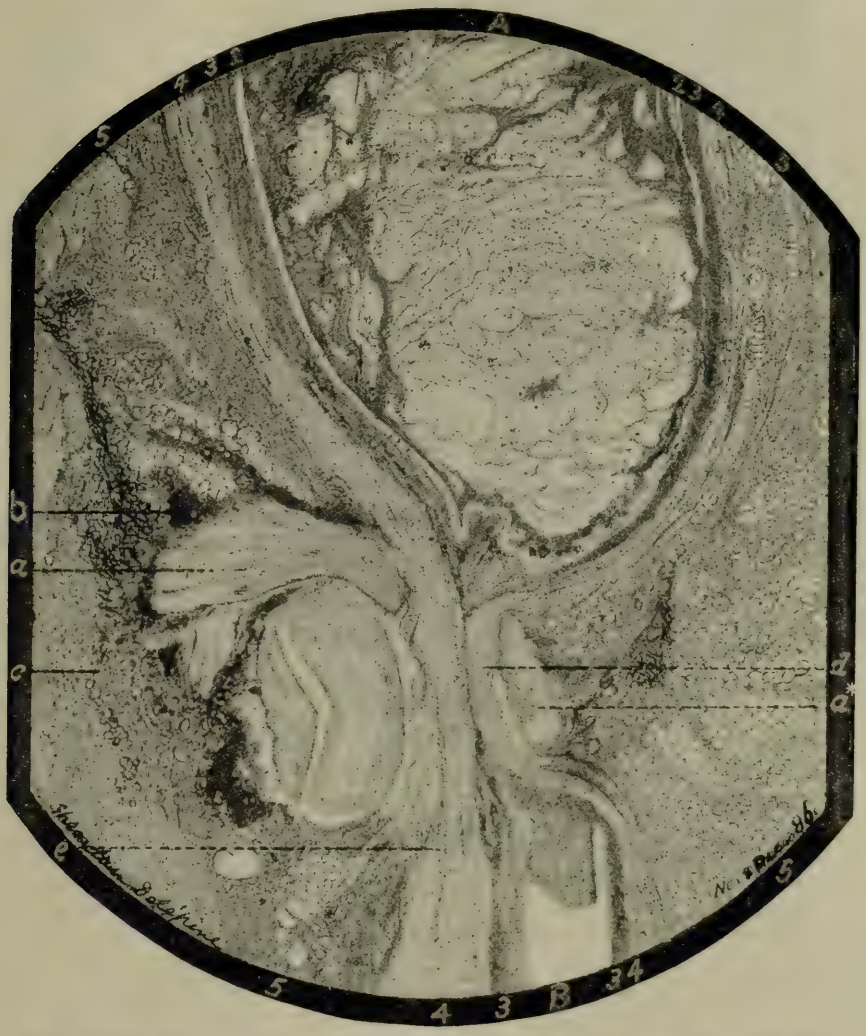

Fig. 7.—A. Proximal part of the artery with the distal part of the clot. B. Distal part of the artery. *a*. Part of the ligature knot. *a**. Ligature on the deep aspect of the artery. *b*. Exudation in the traumatic region. *c*. Proliferative zone around the exudative region. *d*. Adventitia in the constricted portion of the artery. *e*. Infiltrated zone of adventitia. 2. Elastic lamina of intima. 3. Muscular coat. 4. Adventitia. 5. Perivascular tissue. × 16.

FIG. 8.[1]— A. Proximal end of the artery. B. Distal end of the artery. C. First collateral branch above the ligature dilated and hypertrophied. *a*. Ligature on the deep aspect of the artery. *b*. Fissure showing the place where exudation was. *c*. Proliferative zone. *d*. Adventitia in the constricted part of the artery. *e*. Perivascular tissue more vascular than normal. 1. Thickening of the endothelial part of the intima covering the proximal surface of the plug. 1′. Thickening endothelial layer. 1″. Endothelial layer of intima, about normal. 2. Elastic lamina. 3. Muscular coat. 3′. Muscular coat hypertrophied. 4. Adventitia. 5. Perivascular tissue more fibrous than normal, fat absent. 5′. Perivascular tissue. 6. Vascularised older parts of the clot containing still blood-pigment. × 16.

[1] A little more than half of the vessel is represented here in section. A more complete section is represented semidiagrammatically in Fig. 15. This incomplete section has been selected because it showed well the collateral branch C.

with the outer wall of the artery ; the external aspect of the ligature is covered with small cells, extending for a short distance into the surrounding tissues. The artery is much larger above the ligature than below, and contains a large clot, the long axis of which is equal to more than twice the diameter of the vessel. The bulging of the artery is more marked on the side of the vessel which is not supported by the knot than on the other, and on the more bulging side the walls look thinner. The walls are pressed inwards by the knot, and owing to that depression some parts of the walls are cut more obliquely than others ; this accounts, as already explained, for the apparent difference of thickness observed on both sides of the vessel in microscopical sections.

1. *Contents.*

We have already seen that the part of the vessel extending between the ligature and the first collateral branch above (that is, the part in which the blood is stagnant or nearly stagnant) is almost entirely occupied by a clot. This clot is seen on section to be distinctly composed of several parts.

In this case three parts can be recognised for descriptive purposes, viz. a proximal (or most recent), an intermediate, and a distal (or oldest).

(1) *The proximal part* forms the finger-like process which we have already described ; it is paler than the rest of the thrombus, and on opening the vessel was found surrounded by a mass of *imperfectly coagulated blood,* somewhat fibrillated or laminated (see Fig. 1). This loose coagulum was very dark ; it nearly entirely disappeared in the course of preparation of the specimen, only a few masses of altered and deeply pigmented blood-corpuscles remaining around the apex of the clot to indicate, so to speak, that the blood there was not quite normal (see Fig. 10).

The proximal clot itself is composed of several masses separated by thin bands of fibrin, which forms also a thin layer over the greater part of its surface. Near the base of this portion of the coagulum, fibrin is more abundant, and gives that region a somewhat laminated appearance. The most remarkable features of that portion of the clot are the following:

The red blood-corpuscles are little altered, their outline being quite distinct and sharp; but they are paler than normal, with the exception of a few corpuscles forming small additional clumps on the surface of the clot, and having evidently become adherent at some subsequent period.

Leucocytes are seen disseminated all through this proximal thrombus, and are not very much altered. They are more abundant near the surface than in the deeper parts, and in some superficial spots they are remarkably crowded. In some of the latter places the red blood-corpuscles are evidently breaking down.

Other corpuscular elements are also found, but in small numbers in this part of the thrombus; these elements have all the appearances of epithelial or endothelial cells, having a large nucleus, frequently oval, vesicular in appearance, staining less deeply than the leucocytes, and surrounded by an irregular cell body. Some of these large nuclei have very little protoplasm around them. There can be very little doubt that these cells are derived from the endothelium of the artery.

Small granules of doubtful nature are also seen in the clot.

The fibrin, as we have already said, forms bands dividing the clot into several parts, but it is found under other forms too. Thus in many places it forms a fine reticulum passing between individual blood-corpuscles. These fine fibrils may run together so as to form the bands or trabeculæ just alluded to. We may add here that in some places near the surface of the thrombus these fibrinous lamellæ or trabeculæ enclose spaces from which the corpuscles have entirely disappeared. This condition must be

produced either by destruction, escape of the corpuscles, or accumulation of some amorphous material.

Fibrin may present itself under a third form, not less interesting, namely, that of more or less rounded or oval bodies with branching processes extending between the surrounding corpuscles, the long axis of these bodies being generally but not always parallel to the surface of the thrombus. These stellate masses vary in size, some being very little larger than a red blood-corpuscle, whilst the diameter of others is about thirty or forty times that of a red blood-corpuscle. These dimensions are given so that they should not be thought to be simply degenerating white blood-corpuscles, or even groups of them, or of the so-called blood-plates. In the midst of some of these masses red blood-corpuscles can be distinctly recognised, but in others the blood-corpuscles are replaced by a fine granular material, as if they had broken down. In some of the largest of these masses large vacuole-like spaces are found. These masses are most abundant where the ordinary fibrillated form is least abundant. They do not seem to have any direct connection with white blood-corpuscles. Nothing corresponding to blood-plates could be discovered in any specimen.

(2) *The intermediate* part of the clot occupies almost the whole lumen of the vessel; but it is more or less globular in shape, and has a distinct tendency to separate from the walls of the vessel, especially on one side, as shown by the fissures produced in the endothelium and in the superficial parts of the clot itself. The chief differences between this part of the clot and the proximal parts are the following:

The red blood-corpuscles have retained more of their pigment.

The white blood-corpuscles are differently distributed; they have almost entirely disappeared from the proximal superficial parts, whilst they are abundant in the lateral and deep parts. This distribution suggests the idea that having travelled towards the surface many leucocytes

have escaped from the clot where such a thing was possible, but that elsewhere they have remained entangled in the clot.

The endothelial cells are more abundant here, and specially in the lateral portions of the thrombus, where they retain in many places their distinct shape.

The fibrin is more abundant near the lateral surfaces of the clot, where, with the leucocytes and with the endothelial cells, it forms a tolerably thick layer. Near the proximal end the arrangement of the fibrin gives to this part of the thrombus a laminated appearance.

The *third* or *distal portion of the thrombus* is very analogous to the last.

The leucocytes are, however, more abundant in the central region here than in the last case.

The *red blood-corpuscles* are somewhat less distinct, more granular, and darker in colour.

The *fibrin* does not form bands in the midst of the clot; on the contrary, it forms a thick deposit on its surface.

It is evident from the examination of that part of the clot that it must have contracted considerably, remaining connected in several places with the walls of the vessel simply by bands of fibrin. This view is supported by the fact that in the meshes formed by the bands of fibrin at the periphery of the thrombus, masses of red blood-corpuscles are found which are not so altered as those found in the central parts of the clot, and have evidently been more recently coagulated. We should, therefore, add to our list of the three central thrombi a number of small *additional peripheral clots.*

Mode of formation of the clot.—It seems from these observations that the clot is not formed all at once, and that after being formed it undergoes numerous changes due to contraction of its fibrin, migration of its leucocytes, &c.

It will be evident from our description that what we have called the proximal portion of the thrombus is the one that has been last deposited, whilst the distal was the

one first coagulated. We can, therefore, by studying in turn these various parts of the thrombus form an idea of the changes which take place successively in the coagulum.

The first changes which take place after coagulation seem, therefore, to be—

1st. The migration of white blood-corpuscles to the surface. Those remaining behind become altered and degenerate.

2nd. The contraction of the fibrin, which causes the formation of channels between the side of the clot and the walls of the artery, and also in some cases in the midst of the clot itself.

3rd. This contraction leads to two things: A. Into the spaces which result from it fresh blood penetrates, and coagulates in its turn. B. Some of the endothelial cells covering the intima become separated from the subjacent subserous layer, and become embedded in the thrombus (Fig. 9).

2. *Walls.*

Tunica intima (Figs. 9 and 10).—In some places the serous layer cannot be recognised, but this may be due to this layer having been separated from the elastic lamina. This seems to have been due to contraction of the clot. In other places between the clot and vascular walls, and generally clinging to the clot in the form of an external dense layer, a number of round and spindle-shaped cells are found. These cells are apparently partly endothelial cells more or less modified, and partly leucocytes which have come to the surface of the clot.

In other places there is, instead of the intima, an irregular layer of cells, evidently resulting from the proliferation of the elements of that membrane. This is well marked but not excessive at the point of occlusion of the artery, where the intima with the subjacent coats is thrown into folds. It is better marked at some distance above the point of occlusion, and specially at some places

where the proliferated endothelial cells come in contact with tracts of fibrin.

In some preparations another appearance is also very

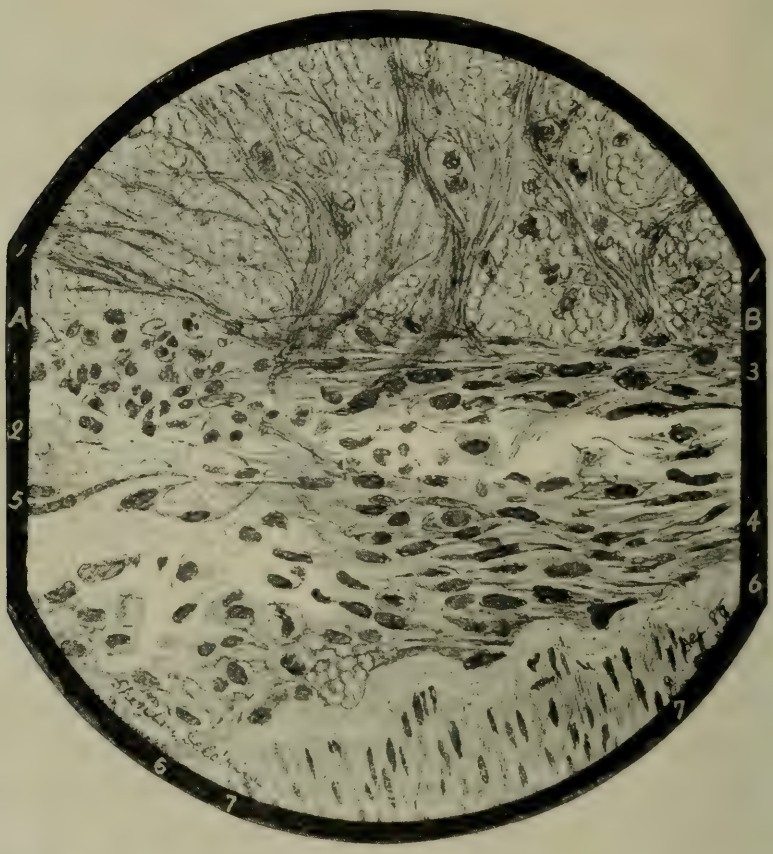

FIG. 9.[1]—A. Proximal end of vessel. B. Distal end of vessel. 1. Clot (red blood-corpuscles and fibrinous bands). 1′. Clot. 2. Fissure into which fresh blood has penetrated. 3. Proliferating endothelial cells of the intima, dragged away from the rest by the contracting clot. 4. Intima thickened by proliferation. 5. Loose endothelial cell. 6. Elastic lamina of intima. 7. Muscular coat. × 400.

distinctly marked. The flat cells of the endothelium, or perhaps of the sub-endothelial layer, have proliferated, and remaining attached by one end, project obliquely into the lumen of the vessel, penetrating more or less deeply into the clot.

[1] This drawing represents, under a high power, the part indicated in Fig. 10 by 1x.

FIG. 10.—A. Proximal part of the artery. B. Constricted part of the artery. *a*. Ligature. *b*. Traumatic zone with exudation. 1x. Tracts of endothelial cells penetrating into the proximal part of the older portion of the clot. 1xx. Endothelial cells of intima, forming layers which split after contraction of the thrombus, and some of which remain adherent to the surface of thrombus. 1xxx. Endothelial layer of the intima, very thin. × 12.

Note.—In Figs. 10, 13, and 15 the outlines are correct, but in order to bring out the intima clearly it has been drawn in black, and in Fig. 10 the contrast is unfortunately not sufficiently well marked.

In the distal cul-de-sac proliferation of the cells of the intima produces a distinct thickening of that membrane, chiefly in the folded region over the ligature.

Muscular coat.—In the neighbourhood of the ligature the muscular fibres begin to atrophy, especially in the region corresponding to the infiltrated portion of the adventitia. In the obliterated portion of the artery there remains a thin tract of tissue representing the muscular coat, and surrounded by the tunica adventitia. A few muscular fibres can be recognised in that region, but the greater part of the muscular coat is evidently torn up at the same time as the tunica intima, and forms with it a convoluted projection into the lumen of the obliterated artery above and below the seat of ligature.

Tunica adventitia.—This is the only coat remaining in its entirety at the seat of ligature.

The constricted portion of the artery measures in length more than half the diameter of the dilated artery above the point of ligature. The structure of the tunica adventitia in this region is distinctly altered. It stains less deeply than in the other parts. It is less cellular, it is less vascular, and the vessels present are almost obliterated by pressure. The cells are elongated, some of them very small, others rather long and wavy. The intercellular substance is coarsely fibrous, probably owing to the compression of the bundles of white fibrous tissue, the elastic fibres remaining unaffected by pressure. The fibres are straightened and rendered parallel, so that this portion of the adventitia looks like dense fibrous tissue with a large number of parallel coarse elastic fibres in its midst.

At the points corresponding to the proximal and distal borders of the ligature, i. e. *in those parts where the pressure of the ligature ceases to exert a direct influence,* a very distinct zone of *cellular infiltration* can be recognised. This zone is very distinct and limited. The cells accumulated in that region are small and irregular in shape.

This cellular zone is easily recognised by the column

of cells separating the fibrous bundles, specially in the outer portions of the coat. Between the most compressed portion of the artery and this cellular region there is an intermediate region where cells seem entirely absent. This indicates that these cells have atrophied, or at any rate that they do not exhibit the usual reactions. The cellular zone corresponds with the point where the ligature ceases to exert its pressure, and where cells of the exudation and infiltrated tissues surrounding the ligature are in contact with the arterial walls.

The adventitia of the proximal side of the artery in the region occupied by the clot is more vascular and cellular than normal. This is true also of the distal side of the artery, but only for a short distance below the seat of ligature.

3. *Perivascular Tissues.*

The vessel is surrounded by adipose tissue in every direction except on the side on which the incision for the purpose of ligaturing has been made. In it there are three important regions to study with relation to the ligature:

A. The traumatic or exudative.

B. The irritative or proliferative.

C. The passive or pressure region.

A. *The traumatic region or tract of incision and laceration* is very limited, and it includes of course not only the path of the knife, but also the track followed by the ligature needle all round the artery. This region is occupied by exudation products, and the products are arranged so as to form *two distinct zones*, which, owing to their relations to the artery, can be called internal and external.

The internal, i. e. the one nearest to the artery or ligature, is composed of small round-cells, which are, apparently, *badly formed leucocytes*, and of the same nature as

those found in the infiltrated portion of the adventitia. This portion covers the external surface of the ligature, and comes in contact with the walls of the artery on both sides of the ligature, and there a few cells wedge themselves in between the ligature and the adventitia as already described.

The external zone of exudation has the appearance of an *ordinary fibrinous exudation*, being composed of a network of fibrin and of leucocytes. Red blood-corpuscles are also pretty abundant (present also in the internal zone, but difficult to recognise among the deeply stained leucocytes). This region is best marked on the side of the knot, and forms there a triangular area, the base of which lies against the small cells covering the surface of the knot, and the apex, directed obliquely towards the surface of the leg, indicating the incision line.

B. External to this mass of exuded material *the tissues are deeply altered by proliferation of their elements.* This layer may properly be called the *proliferative region.* In this region the normal structure of the tissues is more or less altered by proliferation of the nuclei and of the cells; in the immediate neighbourhood of the space filled with exuded lymph the proliferation has been so active that the original structure of the tissues is almost entirely obscured. The fibrous tissue has become entirely cellular, like *embryonic connective tissue;* the *nuclei* of some *fat-cells have multiplied,* and the connective tissue between them, specially along the course of small vessels, has increased in amount; in some places *vessels are seen to be just beginning to form* in the irritated connective tissue. These changes are not distributed equally all round the vessel, but *extend a little further on the side where the knot is;* they also *extend along the walls of the vessel, chiefly on the proximal side,* more than in other directions.

(c) In the most external layer the *tissues are simply altered by the pressure* due to the slight accumulation of products in the two regions just described. This zone

could be called the region of *compression* or *pressure*. The most important changes are those found in some *muscular fibres* which are in a state of *hyaline degeneration*, a condition which may be in part due to interference with the blood-supply.

4. *Ligature.*

The side of the tendon which is in contact with the artery is almost perfectly even and unaltered, but at both ends of the constricted portion of the artery, where the space between the tendon and the coats of the vessels is gradually widening, small cells form a kind of wedge, penetrating between the artery and the ligature (see Figs. 1, 2, 4—8). It is in that region that the coats of the vessel show the small-cell infiltration. *The side of the tendon most distant from the artery* (and therefore in contact with the exudation) *is uneven, being apparently absorbed by the small cells.*

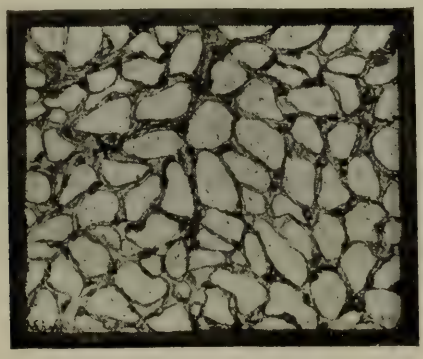

FIG. 11.—Transverse section of a small portion of the ligature five days after the operation. × 400.

The structure of the tendon can easily be recognised (Fig. 11), the cells staining readily, but not quite so deeply as the small surrounding cells. The stains are also more easily retained by the cells of the tissues than by those of the ligature. The fibrous matrix may be coloured very

deeply, but only after prolonged immersion in staining fluid.

The tendinous bands have evidently *a tendency to split and become fissured in the direction of their fibres,* and this occurs chiefly at their periphery. Owing to this, the surface of the tendon is longitudinally furrowed wherever it is in contact with the exudation surrounding it. *The small cells of the exudation penetrate into these furrows* (Fig. 12) ;

FIG. 12.—Longitudinal section through one of the ends of the ligature five days after operation. 1. Bundles of white fibrous tissue. 2. Tendon corpuscles. 3. Exudation composed chiefly of small degenerated leucocytes. 4. Exudation penetrating into superficial fissures of the tendon. × 400.

and where the plan of section is passing quite near the surface of the tendon, either very obliquely or longitudinally, these small cells seem to form long rows separating bundles of tendinous fibres. To understand the true relation of these cells to the tendon it is necessary to exa-

mine a transverse section of a tendinous band. At the points where the small cells are in close contact with it the tendon is evidently undergoing a process of *slow solution or digestion,* but the process is certainly very superficial. Where the ligature is in close apposition with the artery it is less altered than in the parts which are in direct contact with surrounding tissues. This partial absence of changes is more marked on the side occupied by the knot than on the other side, where only one thickness of the ligature separates the coats of the vessel from the inflammatory products.

Ninth Day's Specimen.

General remarks.—The most noteworthy points in this specimen are the extent of tissues involved and the large amount of exudation. The artery is perfectly occluded (see Figs. 2 and 13).

1. *Contents.*

Its *proximal end* is well plugged by a large clot, the long axis of which is equal to twice the diameter of the artery. This plug, although convex, is much flattened; it has contracted and become further separated from the walls of the artery. Organisation and vascularisation are quite distinct along the sides of the artery, and down to nearly the seat of the ligature.

The distal side of the artery does not contain any clot.

The superficial portions of the clot have disappeared, only the laminated base of the proximal part remaining. The rest of the thrombus, although showing traces of more considerable shrinkage than in the last specimen, has apparently ceased to contract. Large channels have, of course, been formed between it and the walls of the vessel.

The clot is distinctly becoming organised, or rather is getting gradually absorbed as newly formed connective tissue encroaches more and more upon it. This destruc-

Fig. 13.—Proximal end of the artery above. Constricted part of the artery below. *a*. Ligature. *b*. Inflammatory exudation in traumatic region. *x*. Distal part of the clot infiltrated with leucocytes. 1. Thickened intima. 1x. Thickened intima penetrating into the sides of the thrombus and passing over its surface. 1xx. Thickened intima splitting owing to contraction of the thrombus. 1xxx. Intima much atrophied in the immediate vicinity of the ligature. 3. Muscular coat. 4. Adventitia. 5. Periarterial tissues. × 12. (See note, Fig. 10.)

tion of the clot is most marked in the more proximal portions of the thrombus. In this region the median parts of the clot have still retained nearly all the characters observable at the fifth day, but in the peripheral portions

there is a mixture of multiplying connective-tissue corpuscles of a more or less fusiform shape, of blood-corpuscles undergoing disintegration (sometimes entirely replaced by small masses of pigment), and of fibrinous débris.

We have said that large spaces are formed at the periphery of the clot, owing to contraction. That such is the case can be easily demonstrated, for in many specimens *fresh coagula*, in most of which fibrin is in great excess, can be distinctly seen *in the midst of the thickened layers of the intima*, and around the much altered parts of the distal portion of the thrombus.

Some remarkable changes can be noticed in the leucocytes found in the various parts of the clot. Some of these cells are much smaller than normal, and contain small granular nuclei, which seem to be the result of some degeneration. Other leucocytes apparently run together to form larger compound masses, in which the original cells can generally be recognised.

Endothelial cells are, of course, now abundant, specially at the periphery of the clot, and in those places where the blood has recently penetrated. This is due to desquamation of the endothelium, and is not special to thrombosis due to ligature (one of us has observed a similar desquamation in many pathological states, and also in the vessels of young animals[1]). In the distal part of the clot there is a considerable increase of small round-cells, and this increase can be easily shown to be due to the penetration of leucocytes from the tissues surrounding the artery, through the walls of the vessel into the thrombus. These cells are therefore of extraneous origin. This is of great importance, for it gives us the means of testing certain views touching the organisation of the thrombus.

If it were true, as Bubnoff asserts,[2] that organisation is

[1] Delépine, 'Trans. Path. Soc.,' 1889, p. 417, &c.

[2] Two recent writers, Ballance and Edmunds, have in the 'Transactions' of this Society, May, 1886, pp. 458, 459, closely followed Bubnoff, and *we therefore disagree with them.* They say, "The microscope shows that a

chiefly due to the passage of migratory cells from vessels surrounding the artery, and from the vasa vasorum, into the thrombus through the walls of the vessel, then we should in our specimen find that organisation spreads from where the leucocytes have accumulated; but we find this is not the case, and that organisation begins in those parts where there is no evidence of diapedesis and very clear evidence of proliferation of endothelial cells. This beautiful experiment, worked out by nature, shows in the same artery the effects of two processes, to which the same results are attributed by different observers, and gives a clear demonstration in favour of those who believe in the essential part played by the intima.

2. *The Walls.*

In the *intima* the proliferation which we had already noticed on the fifth day continues, but the cells take now an arrangement which suggest what is seen when young fibrous connective tissue begins to undergo differentiation. The cells elongate, form tracts which branch in the midst of the organising thrombus, and penetrate even into the parts where the red blood-corpuscles have not yet degenerated. They form thus a distinct network *having all the appearances of a capillary network, but with a few exceptions these tracts of cells do not form as yet tubes through which the blood could circulate. On the convex surface of the thrombus the arrangement of these cells is quite different;* there they are flattened and form several layers, taking the same appearance

cellular infiltration is taking place into the wall of the vessel, around the ligature, and into the clot. . . . By means of these cells the clot near the ligature is decolourised, and, with the ligature, is at last completely absorbed. . . . Riedel is said to have made the inner surface of an artery cohere by multiplication of the opposite endothelial cells without the formation of clot. Of the accuracy of this observation we have grave doubts. Any alteration in the endothelial lining would certainly lead to the formation of a coagulum, and the endothelial multiplication in our specimens is always accompanied by a *leucocytic extravasation*, the latter apparently *being of more importance than the former*," &c.

as the superficial layers of the thickened intima which covers the walls of the vessel. The muscular coat is torn as well as the intima.

The *muscular* and *adventitious coats* are in this case much infiltrated with small cells in the neighbourhood of the ligature, and the specimen shows how easy it is to trace the passage of external products through the walls of the vessels when such a migration is at all important. We would, however, draw attention to the fact that such a migration does not seem to be the general rule, and that in this case we have good reasons to believe that the excessive small-cell infiltration noticeable in the neighbourhood of the ligature was due to some irritant properties of that ligature.

3. *Perivascular Tissue.*

The external aspect of the ligature is covered with a very thick layer of *exudation*, almost entirely composed of ill-formed small round-cells. These cells are evidently perfectly free, and not held by any solid stroma, so that the exudation must have been of a purulent nature. This was noticeable even to the naked eye. It was difficult to bisect the artery without detaching the ligature from the surrounding tissues. The ligature was evidently lying in a small abscess cavity, and was fixed only by its connections with the artery.

The proliferation zone is also very extensive in this specimen ; the fibrous and adipose periarterial tissues are more than half replaced by cellular connective tissue, composed chiefly of small round and spindle-shaped cells with many giant-cells at places. These last cells are chiefly found in the midst of adipose tissue.

4. *The Ligature.*

The ligature is quite distinct, but much more altered than in any of the other specimens, having a tendency to break up into the elementary bundles forming the tendon.

However, the parts in immediate contact with the artery are scarcely altered at all.

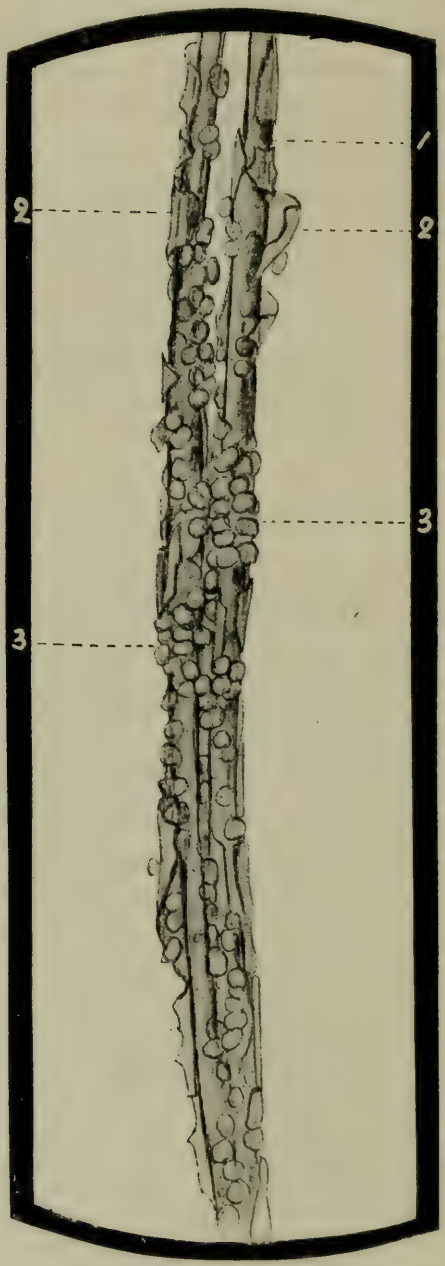

FIG. 14.—1. A tendon fibrous bundle. 2. Exfoliation of the superficial layers of the bundle. 3. Small cells penetrating between the bundles and causing their absorption (phagocytes). Larger cells resembling closely giant-cells or osteoclasts were found in the same situation in some other specimens. × 230.

It is probable that in this instance the tendon was not so perfect as in the other cases. A few mould hyphæ were found among its bundles, so that it is possible that the ligature was not quite aseptic. It is interesting to notice that, under the influence of the small exudation-cells which had penetrated into the spaces resulting from the splitting up of the tendon, the surface of the primitive bundles is altered. The superficial layers of these bundles are scaling off, and this gives to them a worm-eaten appearance. These observations seem to indicate very strongly the importance of homogeneity and perfect asepticity of the ligatures used, but they also show that a certain amount of irritation may be set up without any serious results; indeed, the external appearance of the wound did not indicate in this case that there was any imperfection in the ligature; and, but for the small-celled infiltration of the clot, there is apparently no indication either of the obliteration of the vessel being interfered with.

Thirteenth Day's Specimen.

General remarks.—The occlusion is complete. The two ends of the vessel are nearly of the same diameter, both being rather large; a plug occupies a small portion of each cul-de-sac quite close to the ligature. These plugs had not to the naked eye the appearance of clots. The collateral branch is not more than $\frac{1}{16}$ inch above the seat of ligature (Fig. 3).

1. *Contents.*

The clot has been replaced on the *proximal side* by a mass of fibro-cellular connective tissue, evidently continuous with the serous coat of the vessel (see Fig. 13). This mass of tissue is separated from the *muscular coat* of the artery by *the elastic lamina,* which is still very distinct. Its free surface is very concave. In the deep parts a very few granular masses of reddish-brown pigment can be

recognised. This indicates that a small amount of coagulation must have taken place. The depth of the "organised clot" is less than $\frac{1}{16}$ inch on the side corresponding to the collateral branch. In the *distal side of the artery* there is a smaller mass of newly formed tissue, having exactly the same shape and relations to the walls of the artery as the plug just described. These two plugs seem to be chiefly due to a thickening of the intima.

2. *Walls.*

At the *seat of constriction* both the inner and the middle coats are absent, as in the previous specimens, above and below this point.

The intima becomes gradually thicker towards the ligature, and is distinctly continuous with the laminated cellular layer covering the concave surface of the plug. The deeper layers are much looser in structure, and, quite near the seat of ligature, contain a few brownish-yellow granules of pigment (the remains of a small absorbed clot). In this deeper tissue young vessels are seen, and some have quite a distinct lumen, but they contain no blood.[1] They extend as far as the elastic lamina, but there is no indication of any passing through that membrane. A few seem to be continuous with somewhat wider channels opening on the concave surface of the plug, but this appearance is so rare that in most sections the proximal part of the clot seems to be non-vascular. The distal plug is undoubtedly due to a thickening of the intima; it contains no pigment, but has otherwise the same structure as the proximal.

The muscular coat is much altered near the ligature; the muscular fibres are atrophied, the interstitial tissue increased. Many small, badly formed cells are also found in this region. All these changes are evidently the indications of what may be called a *traumatic interstitial*

[1] *Lumen* here and in the following pages, when spoken of with reference to vessels containing no blood, is not meant to indicate an empty cavity, but that of a clear axial substance.

leiomyositis. They are more marked on the proximal than on the distal side.

The adventitious coat exhibits changes very similar to those described on the first specimen.

3. *Perivascular Tissue.*

The exudation surrounding the ligature has very nearly disappeared; only a very few leucocytes, not even forming a continuous layer, remain on the surface of the ligature. The place of the exudation has been taken by the young round and spindle cells resulting from the proliferation of the periarterial connective-tissue corpuscles. These elements form now two distinct zones.

1. One in *immediate contact with the ligature,* and composed of small round-cells, spindle-cells, small giant-cells, a few fibres, and small vessels beginning to form.

2. Another, *more external and narrower,* but more extensive, composed of large and small giant-cells, large round, spindle-shaped, and irregular cells with large nuclei, bundles of fibrous tissue, and a few fat-cells undergoing atrophy or retrogressive changes. The tissue forming that region has nearly the characters of myeloid sarcoma. This embryonic tissue extends in the direction of the incision, and is therefore more abundant on the knot side than on the other. *A few hairs,* evidently accidentally enclosed in the wound, are found unaltered and embedded in the new tissue, apparently giving rise to no special changes.

Some of these hairs are covered with a distinct layer of small cells.

4. *Ligature.*

The ligature itself is practically unaltered; its *external surface* is hardly more uneven than in the first specimen; the forming young vessels noticed in the inner part of the proliferative region hardly reach the surface of the tendon.

Seventeenth Day's Specimen.

General remarks.—In this specimen, as in the ninth specimen, there are very marked inflammatory changes. The occlusion is perfect (Fig. 4).

1. *Contents.*

Both the proximal and the distal portions of the artery are occupied by *vascular young connective tissue.* Groups of decolorised red blood-corpuscles can be recognised in the proximal plug, which is evidently taking the place of the original clot, which has now almost entirely disappeared.

The proximal plug is composed, as in the last case,—1st. Of a non-vascular layer of flattened cells lining the free concave surface of the plug, and continuous with the endothelium of the thickened intima. 2nd. Of a deeper part occupying the rest of the proximal cul-de-sac, and composed of bands or ramifying tracts of spindle or branched cells. Most of these tracts are young vessels and have a distinct lumen, but as yet contain no blood. Between these tracts small clumps of decolorised and very indistinct red blood-corpuscles and also granules of pigment are found. These are the only remains of the original blood-clot.

In the distal plug (which in this case is larger than in any other case) the thickened tunica intima is continuous, as in the proximal plug, with the new connective tissue. This connective tissue presents the arrangement described above; the young vessels do not, however, present so distinct a lumen, and the tracts of cells are separated by a hyaline material which contains neither red blood-corpuscles nor blood-pigment.

Although the free superficial layers of both plugs are non-vascular, generally speaking, a few pretty large vessels extend through them in or near the axis of the vessel. The meaning of this arrangement will be clear

after the description of the twenty-third day's specimen has been given.

2. *Walls.*

The infiltration of the arterial walls is very marked, chiefly on the distal side. The changes in the *intima* have been described above. The *muscular fibres of the media* are much atrophied ; young vessels begin to extend into the degenerated parts, both from the intima and the adventitia. The *adventitia* is much infiltrated with cells in the neighbourhood of the ligature.

3. *Periarterial Tissues.*

The ligature is still partly covered with *exudation* products, but in many places the *proliferative zone* is now in contact with it. This zone is much more extensive in this case than in the last, and in that respect the specimen bears a strong resemblance to the ninth day's specimen. The adipose and fibrous tissues are being replaced by young connective tissue as in the last specimen. This process is extending to the muscular tissue in the neighbourhood, which shows very well the lesions resulting from interstitial inflammation.

4. *Ligature.*

The ligature itself is more altered than in previous specimens with the exception of the ninth day. The tendon seems to undergo a partial process of slow solution wherever it comes in contact with the small cells surrounding it, and penetrating into the fissures or spaces existing in the tendon itself or between the twisted portions of the knot.

Twentieth Day's Specimen.

General remarks.—There is nothing special in this specimen, which in many respects is similar to the following one. The occlusion is perfect (Fig. 5).

The inflammatory tissue changes are comparatively slight. The line of incision is occupied by cicatricial tissue composed of spindle-shaped cells. The proliferative region surrounds the ligature, which is thus encapsulated. *But there is apparently no adhesion between the new tissues and the ligature, which can be easily separated from them.* The giant-cells are rather less conspicuous in this case than in the ninth and thirteenth days' specimens. The most striking feature of this stage is the *abundance of evidently newly formed vessels in the periarterial tissues.*

Twenty-third Day's Specimen.

General remarks.—The occlusion is complete. Most of the changes which we have to describe now can also be recognised on the twentieth day; but the study of the artery on the twenty-third day is advantageous, as by that time the absorption of the clot is completed, and the beginning of the true vascularisation of the plug of fibrous tissue replacing the clot has become evident (Figs. 6, 8, 15).

1. *Contents.*

The proximal side of the artery is occupied by a plug of fibro-cellular tissue; the length of that plug is not greater than the diameter of the artery. The surface is distinctly concave, almost infundibuliform, and on one side is flush with the opening of a collateral artery, which is evidently much dilated. This plug is composed of two distinct parts, a superficial and a deep part. The superficial, which is continuous with the thickened intima of the

patent portions of the artery, is not vascular, with the exception of a few vessels passing to the deeper layer.

The *thrombus* itself is now represented by a few small masses, a brownish-yellow pigment lying in the midst of the newly formed connective tissue forming the deeper part of the plug, specially in those parts where there is much clear interstitial substance between the newly formed vessels. A few dark masses of different appearance remain also in the most distal part of the clot; and, although their nature is doubtful, it is probable that they are simply remains of partly absorbed masses of fibrin.

The clear material, forming a kind of ground-substance between the tracts of cells representing embryonic vessels, seems to be in part a product of the destruction of blood-corpuscles and fibrin. The masses of pigment above described have a special distribution, which will be better understood after considering the intima.

2. *Walls.*

At first sight the *tunica intima* seems to have simply become thickened, and that more in the immediate neighbourhood of the ligature, and less at some distance from it, causing thus the lumen of the occluded artery to take an infundibuliform shape. This of course goes entirely against what we have described in the previous cases, and seems to support the views of certain writers.[1] The meaning of the appearance can, however, be easily arrived at by a consideration, 1st, of the form and degree of organisation found in the various parts of the organised thrombus; and 2nd, of the distribution of blood-pigment in it (Fig. 15).

The proximal depressed surface of the plug is seen to be formed by several layers of flattened cells, in the midst of which branching embryonic vessels are not found. We have seen that such a layer of cells was extending over the convex surface of the clot at an earlier stage.

[1] Cornil and Ranvier,

Beneath that thickened endothelium we find, on the contrary, a somewhat loose tissue, in the midst of which

FIG. 15.[1]—A. Proximal end of the artery. B. Constricted part of the artery. C. Young connective tissue with embryonic vessels in and about the traumatic region. *a*. Ligature. 1. Thickened intima. 1x. Point corresponding to the level of the clot before absorption, and from which the endothelium began to spread over the clot. The acute angle of reflexion has now been replaced by an obtuse angle owing to the collapse of the central parts, following absorption of the clot. 1xx. See explanation under 1x. 1xxx. Original endothelial and subendothelial layers of the intima indistinct in the neighbourhood of the ligature. 2. Elastic lamina. 3. Muscular coat. 4. Adventitia. 6. Indication of a central canalisation of the plug. 7. Pigmented *débris* of clot almost entirely replaced by young connective tissue with embryonic vessels. × 12. (See note, Fig. 10.)

[1] This section is slightly oblique, and does not pass through the axis of the vessel (which was tortuous) except at the point of constriction, so that the coats a little above the ligature look thicker than if they had been cut mesially.

numerous embryonic vessels with a distinct lumen are seen branching; and this form of tissue we have also seen, at an earlier period, to take gradually the place of the deeper parts of the thrombus.

As to the pigment, we find it only in this loose tissue, both on the deep aspect of the dense layer of flattened cells described above, and in the more distant parts of the thrombus. We come, therefore, to the conclusion that the layers of flattened cells lining the funnel-shaped depression at one time were covering the convex proximal end of the thrombus, and that it has been drawn in, so to speak, owing to subsequent absorption of the subjacent thrombus. As this membrane was attached to the sides of the artery, it must remain fixed there, so that only the central parts could give way.

The *muscularis* is now much altered.

The muscular fibres are now in a state of atrophy, and fibrous tissue takes their place. In several points there are indications of vessels penetrating from the tunica adventitia into the muscular coat, and in some places some of the young vessels formed in the organised thrombus seem to pass beyond the elastic lamina and penetrate into the coat.

These changes are most marked in the immediate neighbourhood of the seat of ligature, and have probably given rise to the opinion that the vessels formed in the organising thrombus were due to an extension of the vasa vasorum into the clot through the tunica media and the intima.

The alterations of structure of the adventitious coat described in relation with the fifth day's specimen are almost quite as distinct in this case. There is, however, a marked increase of vessels. Some of these vessels are evidently enlarged and hypertrophied vasa vasorum, but a great many others are newly formed vessels extending from the periarterial proliferative zone.

The constricted portion of the artery is composed, as in the first specimen, of very little more than the external

fibrous coat of the artery, no distinct trace of the muscular coat being found in the constricted portion of the artery.

3. *Periarterial Tissue.*

There is no trace of *exudation* around the ligature, but the cells of the *proliferative region* are now in close contact with the tendon, and in the *most external portions of the knot many large giant-cells are found.* However, the new tissues are not connected structurally with the ligature. The only place where the ligature seems to be at all firmly connected with the tissues is where it is in contact with the walls of the constricted artery. Even then the adhesion seems to be due to the effects of pressure, for no indication of any inflammatory reaction is found in this portion of the arterial walls (in any specimen), and on the other side there is no trace of any alteration in the ligature itself.

The proliferative region is now rather small and well defined; fatty tissue has disappeared from that region, which is now made up of round, spindle, and giant cells. The giant-cells are now found all through that zone, and many of them are lying against the ligature in the same way as osteoclasts might lie against bone trabeculæ (they are found all through this tissue of new formation). This seems to indicate that they are results of the irritation of cells. They are quite comparable to phagocytes, and all the cells of the same nature which have been described under various names by various authors during the last forty years. *In the midst of the proliferative zone, which therefore must have filled up the space originally occupied by the exudation*, portions of hairs and even of sponge are found; they are embedded in the new cellular tissue, and do not seem to cause much disturbance by their presence. The giant-cells are apparently a little more numerous in their neighbourhood, and the

spindle-shaped cells are arranged more or less concentrically around them.

The most important features of this specimen are certainly increased vascularity of the periarterial tissues, and the distinct evidences there are of an extension of the periarterial and intra-arterial embryonic vessels towards each other, this process evidently leading to the establishment of a new set of vessels through the degenerated portions of the walls of the obliterated vessel.

4. *Ligature.*

The ligature in all the other cases is in perfect contact with the adventitia in the constricted portion, and when it is in contact with the artery it does not seem to have undergone any change. On its outer aspect it is in many places somewhat irregular, but on the whole the ligature is in this case much less altered than in some of the precedent cases.

V. Summing up and Interpretation of the Changes observed.

The operation for ligation, when completed under the conditions described in this paper, results in—

1st. The presence of a *foreign aseptic body* round the walls of the artery at the point of constriction.

2nd. A *solution of continuity* in the periarterial tissues.

3rd. The *laceration* of the two inner coats of the vessel.[1]

[1] This solution of continuity, which in healthy arteries is obtained by using a definite amount of strength, must occur in diseased arteries under very different degrees of pressure, according as the inner coat is more or less brittle and the middle coat contains more or less elastic elements. The larger a vessel the less is its middle coat liable to rupture. The more atheromatous and calcified, the more are the middle coats likely to give way, even before the artery is completely constricted. Admitting that it were judicious to tie a calcified artery, it is evident that the calcified plate found in the deeper

4th. The *compression* of the external coat.

5th. The complete *obliteration* of the lumen of the artery and the production of two culs-de-sac, a proximal and a distal, which have the same walls as the vessel itself, even in the region where the inner coats are ruptured. This effect is brought about by the contraction of the muscular coat, and possibly to a certain extent by the elasticity of the elastic lamina, which leads to closure of the openings of the divided intima and muscular coats. In vessels of the size used in our experiments the contracting coats do more than meet; they become heaped up in the axis of the vessel, so as to form a small projection into the cavity of the artery on both sides of the constricted region; this makes occlusion doubly safe. The opposite sides of the adventitia being brought into close apposition by the pressure of the ligature, the fissure indicating the cavity of the vessel can be entirely discarded. Such is the state of the artery immediately after tight ligation.

The effects of the operative lesions just described are the following:

A. 1st. *A clot* forms in the proximal cul-de-sac, and sometimes in the distal one also.[1] The proximal clot extends as far as the first collateral branch above the liga-

parts of the intima would break and tear the remainder of the intima long before the lumen was completely obliterated. It is, therefore, evident that the tearing of the inner coats, which we describe here as the result of a method of ligation, would occur, whether it were desired or not, in the case of many diseased vessels.

[1] We know, from the experiments of other observers, that when an artery is tied with a loose ligature, even when the lumen is apparently entirely occluded (without rupture of the inner coats), a thrombus forms both on the proximal and distal sides of the ligature. There is, therefore, apparently some marked difference in the extent of thrombosis occurring after tight and after loose ligation. It is to be noticed, however, that in the latter case a thin strand of coagulated blood extends between the proximal and the distal thrombi, and from what we know of coagulation it is quite likely that so long as there exists a tract of blood between the two culs-de-sac, thrombosis beginning in one of them would extend to the other through that intervening tract.

ture. It is not formed all at once; coagulation evidently begins at the point where the coats are divided, and then extends gradually (though not without intermissions) as far as the collateral branch, and even beyond it on the side of the vessel where the current of blood is slowest.

2nd. Whilst the clot is formed within the vessel, lymph and blood fill up the gaps produced by the knife and the needle, and coagulate also.

3rd. All the parts of the vessel compressed by the ligature are rendered bloodless and incapable of vital reaction. This is a result of pressure, and would occur whether the inner coats were or were not divided.[1]

B. The lesions just described lead to further changes in the walls of the vessel and in the periarterial tissue.

1st. *Proliferation of the cells of the intima* near the seat of ligature and as far as the clot extends; the amount of proliferation is, however, not uniform, and depends on several circumstances. *Thus, when there is no thrombosis, it is at its maximum near the place of constriction,* rapidly diminishing towards the heart or towards the extremities. When, on the other hand, *there is a thrombus* occupying the whole lumen of the vessel, and therefore preventing the access of fluid blood to the endothelial surface of the intima, the proliferation is soon arrested in the parts thus covered, and attains its *maximum near the surface of the clot which is bathed by the circulating blood.* As the proximal cul-de-sac of the artery is after tight ligature always occupied by a clot, except when the ligature has been applied just below the opening of a collateral branch of some size, the growth of the intima takes place mostly at the point of junction of the proximal surface of the clot and of the walls of the vessels, and therefore at a distance from the seat of ligature equal to the length of the clot itself. *When coagulation is incomplete,* and an axial thrombus is formed, *pro-*

[1] If, as alleged, the occluding ligature actually causes greater pressure than the tight ligature, this effect of pressure would still more interfere with the nourishment of the walls when the loose or occluding ligature is used.

liferation of the cells of the inner coat *takes place near the seat of ligature* as if there was no thrombus at all.

2nd. Inflammatory changes of the same kind but of much less importance occur in the connective-tissue elements of the *adventitious coat* on both sides, and in the immediate neighbourhood of the ligature, and also to a still lesser extent in the *muscular coat*. These changes seem to be generally slightly complicated by an exudative infiltration. The small cells (leucocytes) forming this exudation sometimes penetrate the clot in great numbers, but they have, as we have shown, no share in the organisation of the clot, and should not receive the misleading name of plastic exudation.

3rd. In the *periarterial tissue* inflammatory proliferation also occurs all round the traumatic region.

c. All the changes which we have described so far are already completed or have distinctly begun before the fifth day after ligature. The following changes result from the extension of the elements resulting from the proliferation of the intima and periarterial tissues into the decaying clot, arterial walls, and the periarterial inflammatory exudation, which undergo gradual absorption.

The intima extends over the surface of the clot and penetrates into its sides. At the same time the clot shrinks, and its proximal part is rapidly absorbed by the young connective-tissue corpuscles invading it. The distal parts of the clot are less altered at first. The endothelial cells covering the surface of the clot form a dense stratified layer; the deeper cells proliferate so as to form tracts of spindle-cells; these tracts are young vessels. The whole clot is permeated by such a network before the end of the second week after ligature. After this it seems probable that the artery can be considered as safely and permanently plugged. The clot is getting more and more absorbed, and its proximal surface sinks in, with its lining of endothelial cells. This surface is at first concave, then funnel-shaped, and thus becomes more and more approximated to the walls of the vessel. This superficial

layer is separated from the muscular coat by the deeper connective tissue containing young blood-vessels, which have now a lumen though they contain no blood. Ultimately these young vessels continue to extend by multiplication of their cells through the muscular coat, and meet those extending from the tunica adventitia and the periarterial tissue. All this is clearly indicated at about the end of the third week.

D. We have made it clear that we did not believe in the existence of a true circulation in the clot for at least three weeks after ligation. We admit that fresh blood penetrates into fissures produced by contraction of the clot, but we contend that this is not equivalent to a true circulation, and we speak of it as a *vacuolation* or a *false vascularisation.*

In the course of the second week after ligation embryonic vessels become evident in the tissue resulting from the proliferation of the intima, as well as in the periarterial inflamed tissues. In the clot, however, these vessels, which are at first solid, even when they have a distinct lumen, cannot serve the purposes of circulation before they communicate with other vessels which may serve as outlets. These vessels, although temporarily useless, are nevertheless vessels, and the *vascularisation* at this stage might be called *potential.*

After the third week, owing to the inosculation of intra- and extra-arterial young vessels through the gradually degenerating muscular coat, circulation becomes possible, so that *effective vascularisation* can be said to exist after that date.

E. *The ligature* has during the whole of this time got gradually but very slowly absorbed. At first it was embedded in the exudation products. After decay and absorption of these the inflamed periarterial tissues come in direct contact with it, and form a distinct capsule for it. By the end of the third week the kangaroo tendon ligature practically supports the walls of the vessel as efficiently as on the first day.

F. The changes which follow are only indicated in our specimens. They are—

1st. Contraction of the newly formed connective tissue, causing obliteration of the old vessel.

2nd. Complete absorption of the ligature, and probably of all the parts of the vessel which have died from the interruption of their blood supply by the pressure of the ligature or interference with the vasa vasorum.

3rd. Dilatation of the large collateral vessels above the seat of ligature.

4th. Owing to the formation of vascular connective tissue round the ligature a certain amount of circulation may be re-established between the proximal and the distal ends of the ligatured vessel through the small newly formed vessel.

5th. The vasa vasorum of the distal part of the vessel may also be supplied with blood by anastomosis with small branches derived from the collateral branches of the artery above the constriction.

Hogson had formerly remarked that at first, *i. e.* before the complete dilatation of the collateral branches of some size, circulation is carried out by innumerable small vessels, and that the dilatation of the smallest was more marked than that of the largest.

All the changes we have just described are those which can be observed in a medium-sized healthy artery. We insist upon the fact that we have not thought it desirable to introduce into this investigation a consideration of the effects of the size, situation, age of arteries, or of the species of animal used for experiment. But by comparing our results with those of other observers who have also applied tight ligature to various healthy arteries of several animals, such as the dog, the sheep, the horse, and even man, we have been able to satisfy ourselves that our practical results were in agreement with those of our predecessors. Our chief aim now, however, is not so much the discussion of the question in all its practical details, as the consideration of the nature of the processes which lead to the occlusion of a vessel the inner

coats of which have been divided by ligation. These results are comparable only with those obtained *on similar bases by other methods*, such as, for instance, those which do not imply division of the inner coats. The discussion that follows must therefore be considered as one of principle, and not as one of details. We, however, know from a careful study of the subject that the principles which we support, and which have been supported by the great majority of surgical authorities since the time of Jones, are more generally applicable in practice than the opposite principles. Our investigation seems to us to give a large number of physiological facts in support of this view. It must be remembered that we have had all these facts before us for more than five years, and we have seen no reason to withdraw any of the statements made by one of us in 1887,[1] and in which we both concur.

VI. Criticism of Views held by other Observers.[2]

1. *Anatomico-physiological.*

The pathologists and clinical observers who have studied the subject from this point of view have devoted the greater part of their attention to the ways in which, 1st, the temporary mechanical obliteration of an artery brings about the *permanent organic transformation of the vessel into an impervious cord;* 2nd, the *interrupted circulation is re-established* by new channels; 3rd, the *ligature is either expelled* from the tissues or *destroyed.*

Many other points have been discussed especially by

[1] Delépine, 'Brit. Med. Journ.,' Aug., 1887.

[2] In this discussion we will not allude to observers who have written during the last decade, except for the purpose of discussing some practical points. The views which we advance here regarding the changes occurring in the thrombus and walls of the vessel are *exactly the same* which one of us had already brought forward in 1887. They have since been confirmed by other observers. They differ considerably from the views then supported by Mr. Ballance and Dr. Edmunds.

practical surgeons, but the three just mentioned are those on which, in principle, the operation can be said to rest.

The first writers, such as John Hunter, Jones (1805), Hogson (1815), Stilling (1831), Hasse (1846), Rokitansky (1852), Weber, Billroth, &c., seem to have studied chiefly the contraction and retraction of the divided coats, and the phenomena evidenced by the clot, such as the arrangement of its layers, its vascularisation, its regressive changes, &c. The clot was generally supposed to take a part quite *sui generis* in the process of occlusion. The fact that the ligatures were allowed or desired to ulcerate their way through the coats of the vessel brought about changes of a special nature. The destruction of the coats of the vessel at the seat of ligature led to the formation of a mass of young connective tissue, which rapidly took the place of the destroyed portion of the artery, and the production of new vessels in that region did often bring about results which were common subjects of discussion among the pathologists and surgeons of the day. Inasmuch as the suppuration and ulceration led to the destruction of a portion of the vessel, the formation of *extensive plugs* above and below the seat of ligature was desirable to prevent the occurrence of secondary hæmorrhage. Hence the importance attached to the formation of the thrombus, to the retraction and contraction of the divided coats, to the distance of the ligature from the first collateral branch above, &c.

After the introduction of the antiseptic method and of aseptic ligatures, the conditions of the operation became so altered that some jumped to the conclusion that everything occurring after ligature under the new conditions must differ wholly in principle from what had been previously supposed to be the case. This phase of opinion, favoured by the very narrow view which Cohnheim had taken of inflammatory processes, led to the publication of ideas such as those of Senftleben (1879), the chief merit of which has been to create adverse criticism and to lead to further experiments.

Despite the passing fashions resulting from the injudicious application of true principles, the knowledge of the changes occurring in vessels after ligature has been growing slowly, owing to the patient and sound work of the observers already mentioned, and also more especially of Virchow (1862), Waldeyer (1867), Bubnoff (1867), Thiersch, Cornil and Ranvier, Durante (1872), Zahn (1875), Pitres (1875), Riedel (1875), Quincke (1876), Schultz (1877), Auerbach (1877), Baumgarten (1876–7), Tillmanns (1879), Shakespeare (1879), Raab (1879), Foà, Pfitzer, Arnaud, Szuman, Hamilton (1881). During the last decade several other workers have continued the work begun by those just mentioned. Many other names might be included in the list.

A. *Coagulation.*—Virchow and Rindfleisch attributed the formation of the thrombus chiefly to stagnation of the blood.

Brücke (1857), Lister (1863), Zahn (1875), Cohnheim, and others have, on the contrary, insisted on the importance of the alteration of the endothelial surface.

Baumgarten and Senftleben have tried to prove that in the absence of septic changes coagulation does not take place.

These different views have been more or less discussed by various writers, but we may consider them briefly in the light of our own experiments.

Our six arteries were tied by a skilful operator; the ligatures were perfectly aseptic (with perhaps one exception); the wounds healed without suppuration and by primary intention; yet in all cases well-marked coagulation occurred. We feel, therefore, convinced that the theory of Baumgarten and Senftleben concerning the absence of coagulation when aseptic ligature is used cannot be maintained, and on this point we fully agree with Arnaud.

The shape of the clot, its extension along the side of the artery opposite to that of the collateral branch above the point of occlusion, &c., all favour the view that stag-

nation must have at least a share in the causation of thrombosis. A difficulty, however, is raised by the absence as a rule of a coagulum in the distal cul-de-sac, where the blood must be nearly as stagnant as in the proximal cul-de-sac of the artery.

The same difficulty occurs when Brücke's views are considered. The lesion of the intima is practically the same on both sides of the ligature, and yet in our specimens permanent coagulation is the rule on the proximal and the exception on the distal side. Between the two the evidence, so far, is apparently in favour of the old stagnation theory. We believe, however, that both theories are partly correct, but that the endothelial lesions are really started by deficient nutrition due to stagnation. We suspect that the conditions of *circulation in the vasa vasorum* are not the same above and below the ligature, but it would be premature to discuss this view, as we can adduce no experimental proof in support. It is, however, important to remember that when double ligature is applied to a vessel no coagulation takes place in that vessel between the ligatures for some considerable time. Simple stagnation is therefore not enough. We are thus driven to consider all the theories we have discussed as incomplete. A factor which has not been considered yet may, perhaps, supply the explanation which has yet to be found.

When a fluid containing solid particles circulates in a vessel into which blind appendices open, the conditions of pressure and of distribution of solid particles are not the same in appendices having different directions and openings of various sizes. We only indicate here a differential element, but the discussion of this question would carry us beyond the limits of this paper.[1]

[1] That a difference exists between the state of the blood in the proximal and distal culs-de-sac seems to be clearly indicated by the fact that when the vessel is contracted without the inner coats being torn, coagulation (according to Mr. Ballance) is always found on both sides of the ligature. As this is not the case with tight ligature with which rupture of the coats is obtained, it is to be supposed that when loose ligature is used the process of

Our *first conclusion* is, therefore, that the *occurrence of thrombosis after ligation is not determined (though it may be modified) by septicity; that it is partly due to stagnation of the blood, and to certain conditions of circulation above the seat of ligature, and secondarily to degenerative changes in the endothelial lining of the vessel.*

B. *Organisation of the thrombus.*—Putting aside the view supported by Weber, Billroth, and others, that the clot may become entirely or in part transformed into some permanent living tissue, there remain two antagonistic theories to consider.

Bubnoff (1867) sought to prove that the *organisation of the clot was due to the immigration of leucocytes escaped by diapedesis from the vessels around the artery.* Schultz (1877), Senftleben (1879), Ziegler (1889), and others have supported these views by further experiments.

On the other hand, Waldeyer (1867), Cornil and Ranvier, Durante (1872), Riedel (1875), Baumgarten (1876), Shakespeare (1879), Raab (1879), and many others have supported the view that the *proliferative changes occurring in the intima were the most important factors in the organisation of the clot.*

The facts which we have been able to observe make it impossible for us to agree with Bubnoff and those who think with him. We cannot even accept as satisfactory the conciliatory hypothesis advanced by some, such as Pfitzer and Ziegler, and quite lately Ballance and Edmunds, who seem to imply that both the processes may play an important part in the organisation. We share the views of Cornil and Ranvier and Waldeyer, so well supported by the experiments of Durante, Riedel, Shakespeare, &c. We, however, take exception to the statement made by Cornil and Ranvier and their followers regarding the region where the proliferation of the intima is most marked. These observers think that the proliferation is most evident in the neighbourhood of the

coagulation extends from the proximal to the distal part of the vessel through the constriction.

ligature. We found, however, that this was the case only (the inner coats being ruptured) when there was little or no thrombosis.[1] When there was a marked clot, the *proliferation of the cells of the intima was most marked at the level of the proximal end of the thrombus*, and therefore separated from the ligature by a space equal to the length of the thrombus. Shakespeare has noticed in one of his experiments a marked thickening of the intima at a certain distance from the ligature, but under the influence of Cornil and Ranvier's views he tried to explain this by supposing that he had held the vessel with forceps at that place. We may add here that we have not only observed the part which the tunica intima takes in the organisation of the thrombus, but can furnish by one of our specimens a demonstration of the passivity of the migrated leucocytes. In our second specimen we have shown how the deep parts of the thrombus were invaded by an army of leucocytes coming from the vasa vasorum and periarterial vessels. These leucocytes were most abundant in that part of the clot which showed little if any sign of organisation, and which by comparison with the other specimen was the very part of the thrombus which disappeared by absorption without being permeated by embryonic tissue. Such a fact, bringing into contrast the two processes, shows clearly that the essential factor in the organisation of the thrombus is the proliferation of the cells of the intima in the parts where they can be reached by the blood.

c. *The vascularisation of the thrombus* has been as great a source of discussion as those questions which we have already dealt with.

Early writers—for instance, Stilling (1834)—described

[1] When there is no or little thrombosis, or when there is only an axial fusiform clot, as is often the case when the inner coats are not ruptured, fluid blood can reach the intima as far down as the place of constriction; this, we suppose, is the only explanation of the difference existing in the seat of proliferation of the intima between tight and loose ligature.

the appearance of vessels in the thrombus without giving any distinct opinion as to their mode of origin. These observations were chiefly based on the study of injected specimens. Stilling noticed that in recent thrombi the periphery was more readily injected, whilst in old thrombi the reverse was the case. From our observations it seems evident that the canals injected by Stilling in recent thrombi were not vessels at all, but simply spaces produced by the shrinking of the fibrin; whilst those injected in older thrombi were the true vessels formed during organisation, vessels which, as we have shown, cannot be efficient, because not channelled, before the end of the second or third week.

Rokitansky (1852) went so far as to say that he had never observed the formation of vessels in a thrombus. Cornil and Ranvier think that the vascularisation of the clot is brought about by the growth of vessels extending from the vasa vasorum into the lumen of the vessel filled by the thrombus. This would take place at the end of the artery where the tunica media has disappeared, and the new vessels would extend within the granulations containing them in a direction parallel to the axis of the vessel, and grow towards the heart.

Hamilton (1881) believes that the extension of the vessels takes place in the form of loops, which penetrate into the clot as they would into a sponge.

Schultz (1877) reverts to the old idea that vascularisation begins within the lumen of the vessels, and evidently admits an independent formation of vessels within the clot, but believes that the organisation depends on the immigrated leucocytes.

Ziegler suggests that at first, at any rate, the vascularisation described by Schultz is not a true vascularisation.

From what we have already said it will be evident that none of the hypotheses just mentioned cover all the facts which we have observed.

We think we have proved—

1st. That in a recent thrombus irregular channels are produced by shrinking, and that the blood penetrating into these channels or spaces coagulates after a time. This *false vascularisation* is the only form of canalisation observed during the first ten days.

2nd. That the proliferating cells of the intima are arranged in the young connective tissue invading the clot in the same way as in other forms of young connective tissue. Branching tracts of spindle-shaped cells are thus formed, and produce the appearance of a vascular network. There is, however, no true circulation through these vessels, most of which have no lumen. This condition may be called *potential vascularisation.*

3rd. That after a time the inflammatory changes affecting the coats of the vessel lead to the formation of young vessels continuous with the vasa vasorum, and extending through the degenerated middle coat towards the obliterated lumen as well as in other directions, and that ultimately the young periarterial and endoarterial vessels thus formed meet and inosculate. It is only after this inosculation that *true circulation* becomes possible. Such circulation does not probably take place in a medium-sized artery before the twentieth day. Now it is evident that when it was customary to let the ligature ulcerate through the coats of the vessel there was for a while between the two gaping ends of the artery a tract of young connective tissue. This tract gradually became continuous with the young connective-tissue plug which had replaced the absorbed thrombi. The blood contained in the vessel above and below the point of constriction was therefore separated by a bridge of vascular connective tissue. We have shown that, as the superficial parts of the clot are rapidly organised while the deeper parts of the clot (which the nutrient fluid cannot reach and where organisation is rather deficient) are absorbed, the surface of the plug becomes gradually more and more concave, and sometimes even infundibuliform. Consequently most of the blood-stream is directed towards

the central portions of the vascular plug, and there is a tendency for the central channels to enlarge. This explains the formation of the central vessels found at various intervals after ligation, which have attracted the attention of Lobstein, Stilling, Blandin, and others. The arborescent sproutings of vessels described by Jones, Ebel, Manec, &c., do not invalidate this explanation. In fact, Stilling observed that sometimes the central vessel did branch. In all the cases just alluded to the partial re-establishment of circulation had taken place very slowly, for the observations relate to vessels which had already been tied for months or for years. This kind of circulation is, therefore, of little practical interest, since, by the time it becomes evident, the usual collateral circulation is already fully established, and the lesions for which the artery had been tied either cured or beyond the reach of treatment by ligation.

It is perfectly evident that when a tight ligature is used secondary channels may form between the two ends of the constricted vessel. Equally patent is the fact that so long as the ligature and the portion of adventitia which it encloses are not absorbed, the new channels must pass round the ligature. Messrs. Ballance and Edmunds have found that seventy-three days after ligation the ligature was not yet entirely absorbed. It is *therefore evident that a direct intermediate central vessel would not be likely to be established before the end of three months when the ligature is applied tightly.* As we have seen, this formation of a central vessel is of no moment whatever unless it occurs so rapidly after ligation as to permit the re-establishment of the flow of blood before the desired effects of ligation have taken place. Messrs. Ballance and Edmunds have shown in their experiments that when a ligature is so loosely applied as to cause only slight constriction of the vessel there is no complete obliteration of the lumen of the vessel forty-four, fifty-eight, and even seventy-three days after ligation; in two of their cases a loose clot existed at the seat of constriction, but such a

clot would be a poor guarantee of obstruction, specially in the case of large vessels and in case of suppuration, as shown by their experiment No. 18.[1]

D. *Fate of the ligature.*—From our observations it seems evident that after the first irritation caused by the operation has subsided, the tendon ligature remains in the tissues as a non-irritating foreign body. Absorption of the tendon goes on slowly, but long before the process is complete a kind of capsule of vascular young connective tissue surrounds the ligature in every direction, that of the vessel excepted. The ligature, therefore, continues to support and constrict the vessel until it is entirely replaced by connective tissue. The process of solution of the ligature takes place wherever the living tissues come in contact with it, and is more rapid where the ligature is split into fibrils or small bands than where it is smooth and forms dense tracts.

E. *Changes observed in the traumatic and irritative periarterial regions.* These are interesting, as they show distinctly the relations which proliferative and exudative changes bear to the healing process. It is evident from our experiments that, however perfect the process of healing may be, there is always a slight amount of inflammatory exudation, specially where, owing to the presence of a foreign body (such as a ligature), parts cannot be brought in absolute contact. This exudation has all the anatomical characters ascribed to croupous exudation; it is composed of leucocytes, red blood-corpuscles, and plasma, from which a network of fibrin soon precipitates. As in the case of other croupous exudations, these products show no tendency towards organisation, but soon degenerate and are absorbed. On the other hand, proliferation of cells takes place actively in the surrounding connective tissue, even the adipose cells showing active signs of proliferative activity. Then the fat is absorbed, the nuclei multiply, giant-cells are formed in the midst of the young sarcomatous-looking

[1] 'Med.-Chir. Trans.,' vol. lxix (1886), p. 455.

tissue, especially round the foreign bodies which are in the wound. In no case has it been possible to find any evidence of the formation of the giant-cells by the coalescence of leucocytes, whilst in many places these cells were found to have the relations of ordinary connective-tissue corpuscles or fat-cells. Gradually, as the exudation is absorbed, this young sarcomatous tissue comes in contact with the ligature, and shows at first embryonic potential vessels, and afterwards pervious or effective ones. The absorption of the periarterial exudation takes place at the same time as that of the clot, or perhaps a little more rapidly, so that the process of organisation goes on at the same time without and within the vessel, and is brought about by proliferation of pre-existing connective-tissue cells.

2. *Clinical.*

Whether it is the best practice to tie an artery, in its continuity, by a tight or a loose ligature, is an old but, as will be recognised, a still debated question. By tight ligature we signify rupture of the inner coats of the vessel, while the loose ligature aims at occlusion by mere compression of the arterial walls. The adoption of the antiseptic system, the possibility of applying an aseptic, absorbable ligature through an aseptic wound, allows us at the present time to survey the whole matter from an entirely new aspect, and the greater part of the existing literature on the subject deals with matters now beside the question, and with conditions which need no longer exist. Even with this state of affairs it is said that loose ligation is the best, the most scientific, and the safest proceeding. To this contention we wholly demur, traversing all the three arguments alleged above in its favour.

Experiments made under the favorable conditions of the physiological laboratory on the perfectly healthy arteries of young animals cannot of course settle such questions,

considered in their practical or clinical aspects. Still they furnish invaluable information, for they illustrate the typical changes taking place in the normal mode of repair. All the knowledge gained by these experiments has, so to speak, to be translated into the language of practical surgery. Viewed in this light, the results of our experiments furnish, to our minds, strong arguments in favour of tight ligation.

For the sake of convenience, and because the various branches of the subject are clearly and exhaustively set forth, we will adopt generally the headings given by Messrs. Ballance and Edmunds in their paper[1] advocating the advantages of the loose or occluding ligature. The case for ligation without rupture of the internal coats could hardly be better stated than it is in this communication.

It appears to be generally conceded that the properties of a ligature are good when the material is uniform in structure and behaviour; in addition it must be strong, unirritating, easily rendered perfectly aseptic, slowly absorbed, and capable of being tied with a close knot which is not liable to slip. Whether kangaroo tendon, ox aorta, whale tendon, or catgut be used matters comparatively little, especially if the ligature is tied tightly. We may say at once, however, that kangaroo tendon seems to us by far the best of the materials enumerated. In the case of loose ligation the quality of the particular material employed becomes at once a question of importance. A defective catgut ligature, for instance, might be too rapidly absorbed, and this defect would be likely to lead to too rapid return of the blood-stream through a loosely ligatured artery. Such a mischance would matter but little in a laboratory experiment. The artery might return almost to the normal state it was in before ligature, or an interesting diaphragm be formed owing to the perforation of the web of proliferating intima-cells stretching across the lumen of the vessels. But if the artery had been tied for aneurism the chance of cure would have

[1] 'Med.-Chir. Trans.,' vol. lxix, p. 463.

been much diminished. It must be remembered that, in practice, when an artery is tied we do not only require to safeguard against secondary hæmorrhage; a distinct object has to be gained, such as the cure of an aneurism or the control of hæmorrhage from some more distal vessel which cannot be secured locally. One of us had recently under his care a case in point. A man, aged sixty, had severe hæmorrhage from a cancerous ulceration in the floor of the mouth. Half of the tongue had been removed six months previously. In three attacks the patient lost 90 oz. of blood, on one of these occasions losing 60 oz. Local remedies proved fruitless. Ligature of the lingual artery was rendered impossible by the presence of extensive cancerous infiltration under the jaw and in the neck. The rather unorthodox treatment was therefore adopted of ligaturing the common carotid. The vessel was far from healthy, and very slight traction on the tendon ligature caused the internal coats to give way distinctly. The wound healed by first intention, and there was no further return of the hæmorrhage.

The more certain a ligature is of occluding the vessel permanently the better is it suited for its purpose. In double distal ligature of the carotid and subclavian for thoracic aneurism the re-establishment of the circulation through the main vessels which had been temporarily obstructed might, even after a long period, vitiate any benefit gained by the operation.

Catgut, being formed of several different tissues twisted together, can never be quite so reliable as a material nor so homogeneous as tendon. The behaviour of catgut when buried in the tissues is also a matter of some uncertainty, and in the case of loose ligation, when it is of extreme importance that the ligature should remain as long as possible in proper contact with the arterial wall, this uncertainty becomes a matter of serious moment. No doubt the defect inherent in catgut is neutralised to a very great extent by chromicising the ligature, but this process renders the material hard

and less adapted to be tied with a properly close knot. We may mention here that although the experiments described above were made with a chromicised tendon, we now think that this mode of preparation is not really of any advantage, and that by impairing the suppleness of the tendon it interferes with its efficiency. *The main point, however, is not so much now the choice of material as the choice of a method of ligation which enables the surgeon to select whatever material he may chance to prefer.* A tendon may, of course, be found defective though most carefully selected and prepared. Still, in our opinion, the risk resulting from a defective ligature, or of one that undergoes too rapid absorption, is less if the first knot be tied firmly, whatever the material used. It has been urged, as an argument against tight ligation, that it is not justifiable to do more than is absolutely necessary to attain the end in view. The epithet is too strong in a matter in which there is much to be said on both sides of the question. To our minds it is dangerous to lay down an arbitrary minimum standard inapplicable to all cases. The very expression "rupture" of the coats appears to shock some people, and to imply that the knot of the ligature is drawn not so much with firmness, decision, and a sense of a distinct end to be achieved, as with a species of surgical brutality. The gentleness and *quasi*-refinement of the one procedure are contrasted with the rough coarseness of the other. But the rupture of the coats will usually occur, whether it is desired or not, whenever these coats are much diseased and stiff and the artery is sufficiently constricted as to cause occlusion. Some margin should be allowed; and the safest practice appears to us to be to do no less than circumstances may possibly demand. Tight ligation allows such a margin, and does so, as we have endeavoured in part already to show, without incurring any extra risk or even disadvantage. We find ourselves unable to subscribe to the opinion that under the antiseptic system it does not matter whether the internal tunics be ruptured or not. We think it is decidedly better to rupture them. Nor will such

evil consequences follow, with tight ligation, if asepsis fails to be maintained in the wound. The best results will be secured when asepticity is complete, and it is of paramount importance to secure such a condition. Still, failure in this respect may occur. Concerning ourselves now only with the local consequences, we should expect that, in a septic wound, the ligature would be much more rapidly consumed or absorbed. But the occlusion of the vessel will have been secured, as our specimens demonstrate, in the most rapid and efficient manner, and the operation, considered in its mechanical aspects, will have had the best chance of bringing about the results aimed at. Tight ligation, therefore, allows a margin, and one that it is very desirable to provide, for possible failure in the endeavour to secure asepsis.

The restoration of the natural channel through the ligatured portion of the artery, already referred to, must always be a matter of uncertainty. If the knot of a loosely tied ligature gave way almost immediately after ligation the lumen of the vessel might be restored intact. But a considerable time must, under more ordinary circumstances, elapse before a diaphragm is formed, or the occlusion, complete for a while, is tunnelled through. Such restoration will only occur after loose ligation. By some this contingency is considered desirable. We hold the opposite opinion. The assumption that the nutrition of a limb, threatened by the ligation of its main artery, would be saved by the restoration of the temporarily occluded channel, is very theoretical. The limb would die if deprived wholly of blood for, say, twenty-four hours. Any portion of the limb would become gangrenous if similarly starved. The period at which the vitality of a limb is most imperilled is during the first few hours after ligature. Subsequently the efficiency of the collaterals becomes more pronounced. The aneurism, if there be one, diminishes in bulk, and the obstruction is thereby lessened. By the time that the lumen of the ligatured vessel could be safely restored without prejudice to the condition for which ligation was

performed, the circulation through the limb is already as perfect as it need be. No good can really come of the reopening of the closed channel. It is much more likely in practice that harm might result. The best imaginable condition after ligature of a main artery for aneurism is for the nutrition to be perfectly maintained by means of the collateral circulation. If this physiological result be secured, there is no need to trouble about the absolute restitution of the anatomical condition. The latter, in fact, would only tend to make the return of aneurism more probable.

In the event of suppuration occurring at the seat of ligature, the method of tight ligation still offers, to our minds, the greater security. One of two conditions may occur. Either an abscess may form round the constricted vessel, the ligature still remaining *in situ;* or, secondly, the loop may cut its way through the vessel, which being thus divided allows the pus to have access between the ends. If the abscess occurs round the vessel, ulceration may take place into it, and lead to fatal hæmorrhage. Specimens abound in museums illustrating this process, one that has been admirably treated of by Syme[1] and many other writers. Now, the more natural the condition of the artery, the greater is the risk of the hæmorrhage resulting from such ulceration, for the opening is made into a vessel in which the full blood-stream is flowing. The ligatured artery is safer than the sound one. The greater the extent of occlusion and the less the blood-

[1] 'Contributions to the Path. and Pract. of Surgery,' 1848. On p. 267 Syme writes:—"As this case (q. v.), together with others of hæmorrhage under similar circumstances, has been referred to in support of the opinion that an abscess may be converted into an aneurism by ulceration of a contiguous arterial trunk, I think it right to remark that although arteries frequently open on the surfaces of sinuses and ulcerated cavities, the only instance of communication with an abscess on record is the one supposed to have occurred in University College Hospital." The meaning probably is that, when ulceration into an artery takes place under the circumstances external hæmorrhage is the common result, and an aneurism is unlikely to form. After an opening has formed into the artery the perforation has frequently been found closed by a coagulum.

current in it, the smaller the risk of grave hæmorrhage. Abscess is not more likely to occur after tight than it is after loose ligation. The occurrence of suppuration is, indeed, a question of septicity, and not of tight or loose ligation. Our specimens show that a good length of vessel—in our opinion more than follows the use of the loose ligature—is absolutely rendered impervious; and it follows, therefore, that the danger from this source after tight ligation is at least no greater than by the other method.

Secondly, it is said the ligature may cut through the adventitia. Experiments and practice appear alike to show that a tightly tied absorbable aseptic ligature does not as a matter of fact cut its way through the vessel, but remains embracing the artery after many days. If the ligature has irritating properties, such as badly prepared catgut may possess, it might work its way through the coats whether it be tied tightly or loosely. The more perfect and more rapid occlusion secured by tight ligature still has the advantage in preventing serious consequences from any such mischance. Or the loop of the ligature may dissolve with undue rapidity, so that the knot with two free ends is set loose. Still the processes set up by the tight ligation render the accident of less consequence than would be the case with loose ligation; for here, again, the more complete and quick closure resulting from the former process offers the best chance of safety under the conditions. Should any of these accidents happen after loose ligation, there is really little save a slightly adherent clot in the vessel to prevent hæmorrhage.

The danger sometimes supposed to result from the tight ligation of arteries in an advanced state of degeneration is, we think, rather theoretical. It must be borne in mind that atheromatous degeneration commences in the deeper parts of the intima, and extends gradually outwards. The altered internal tunics, therefore, would be ruptured with more ease, and in a degenerated vessel might even be divided, when the intention was merely to compress them.

We believe that security is perfectly maintained if these inner coats are cut through. No doubt it is unnecessary to tie the ligature as tightly on a diseased vessel as on a healthy one in order to effect the rupture. But a very large margin is allowed, for with a flat ligature a degree of force that no operator could practically employ would be necessary to cut through the adventitia even in a very rigid vessel. The giving way of the inner coats will be felt distinctly as the knot is closed, and this unmistakable sensation enables the operator to estimate to a nicety the force required. In tying an artery for aneurism it is best to assume that the vessel is in a diseased state. In practice, as far as our researches and experience extend, no evil results have followed tight ligation of diseased vessels with tendon ligatures, and we believe this to be not only safe, but also the best practice. Flat ligatures offer on the whole the greatest advantages. The knot is closer and more compact than in round ligatures, and the shape enables the band to support the vessel more efficiently. When flat ligatures are used, however, it is of importance to bear in mind that the ligature should be drawn tight slowly, and the second knot should also be closed without any jerk.

We wish it to be clearly understood that *we do not dispute the fact that by means of the loose ligature perfect occlusion can be obtained in an artery under suitable conditions.* The results obtained by acupressure gave proof that it was possible to occlude an artery by simply approximating the walls by moderate pressure for a short time. But this practice was but short-lived, for it was found to be inconvenient and of uncertain efficiency. It is probable that many of the arteries tied in a large wound, such as that of an amputation, do not have their inner coats ruptured. Again, attempts were made at one time, though not, we believe, in this country, to bring about the permanent occlusion of an artery in its continuity by means of gently pressing together the walls, a special form of arterial clamp being used for the purpose. This practice, however,

was soon abandoned. Loose ligature is decidedly preferable to any such method. Our contention is that the firm ligature also occludes perfectly, while it is applicable to more—in fact, to all cases; further, it is the safest method when the healthiness of the ligatured vessel is a matter of doubt.

VII. General Conclusions.

I. We advocate the practice of tight as preferable to that of loose ligation, at least as far as general application is concerned,—1. Because it ensures more complete and permanent obliteration of the vessel. 2. It brings about the desired condition with more rapidity. 3. It prevents possible re-establishment of circulation through the vessel. 4. It gives more uniform results. 5. It requires less skill. Any error in the direction of tying the artery with more force than is actually required will lead to no bad consequences. 6. It allows a margin for any possible complications that may arise.

II. The use of an aseptic tendon ligature is recommended —1. Because such ligatures are very uniform in structure and composition. 2. Because being flat they are less likely to injure the external coat of the vessel than a small round ligature, and for the same reason the knot is more compact though not smaller. 3. Because although tendon remains unabsorbed for a great length of time it causes no irritation.

[1] The method was known in Germany as "Arterienklausur." See Billroth's 'Clinical Surgery,' New Sydenham Society's Translation, p. 458.

APPENDIX.

Bibliography.

This bibliography does not pretend to be complete, but gives the sources of statements or views which have been alluded to in the discussion.

No.	Authors.	Date	References.	Title or nature of contribution.
1	Arnaud	1880	Contributions à l'étude de la ligature dans le traitement des anévrismes	Coagulation occurs in vessels tied with carbolised catgut ligature.
2	Auerbach	1877	Dissert. (Bonn)	Ueber d. Obliterat. d. Arterien nach Ligatur.
3	Ballance (C. A.), Edmunds (W.)	1886	Medico-Chirurg. Transactions, 2nd series, li	The Ligature of the larger Arteries in their continuity: an Experimental Inquiry. 1. It is neither necessary nor advisable to rupture the coats of the vessels. 2. The lumen of the vessel must be completely or almost completely obliterated, and the integrity of the walls must be respected. 3. The round ligature, of small size and possessed of certain qualities, is the best (small, round, absorbable ligature).
4	Barth	1837	Presse médicale, p. 62	—
5	Baumgarten	1876	Centralbl. f. d. med. Wissensch., No. xxxiv, Aug. 19	Organisation of thrombus entirely due to changes in intima. Thrombus passive.
	„	1877	Die sögen. Organisation d. Thrombus, Leipzig	Organisation of clot in doubly ligatured arteries in a rabbit.
	„	1886	Berl. klin. Wochenschr., xxiii, p. 385	—

No.	Authors.	Date.	References.	Title or nature of contribution.
6	Billroth	1877	Lectures on Surgical Pathology and Therapeutics, 8th edit., translated by Hackley, New Sydenham Society	Presence of clot in proximal and distal end of large arteries tied in their continuity; inclines to believe that leucocytes contained in the clot play an important part in organisation. Views not very clear.
	,,	1856	Untersuchungen ueber die Entwicklung der Blutgefässe (Berlin)	—
7	Blandin	1840	Journal Hebdomadaire, Mai	Axial vessel formed after ligature
8	Brücke	1857	Arch. für Path. Anat., No. 13, xii; Brit. and Foreign Med.-Chir. Rev.	Stagnation not sufficient cause of coagulation.
9	Bubnoff	1867	Centralblatt, No. 48	Organisation of thrombus due to immigration of leucocytes (doubly tied veins). Vermilion spread in wound found within vessel in leucocytes.
10	Burdach	1885	Arch. für Path. Anat., c, p. 217	Ueber d. Senftlebenschen Versuch., &c. Carotid artery of dead men and rabbits introduced into the abdomen of another animal became filled up with vascular connective tissue, which penetrated into the vessel at the injured ends.
11	Callender	1878	Clinical Transactions, ix, p. 103	Proposal of kangaroo tendon ligature.
12	Cohnheim	1880	Vorles. über allgem. Pathologie	—
13	Cornil and Ranvier	1868	Archives de Physiologie, t. i	Contributions à l'histologie normale et pathologique de la tunique interne des artéres et de l'endocarde. Vascularisation by extension of vasa vasorum into the thrombus. Organisation of thrombus due to neoformation beginning in walls of vessels. Bubnoff's experiment does not succeed with simple ligature.
	,,	1881	Manuel d'Histologie Pathologique, 2nd edit., vol. i, p. 598	

No.	Authors.	Date.	References.	Title or nature of contribution.
14	Delépine	1887	Brit. Med. Journal, ii	Organisation of thrombus formed in an artery after ligature.
15	Dent	1881	Medico-Chirurg. Transactions, lxiv, March	A case of innominate aneurism, &c., with remarks on the behaviour of a tendon ligature.
16	Durante	1872	Archives de Physiologie, p. 491	Sur l'organisation du caillot dans les vaisseaux.
	,,	1872	Wiener med. Jahrb., iii, iv, pp. 143—160	Fallacy of Bubnoff's experiment, in which necrosis of walls of blood-vessels is brought about, unlike what occurs in ordinary ligature.
17	Ebel (von)	1826	De Naturâ Medicatrici sicubi Arteriæ vulneratæ et ligatæ fuerint (Giessen)	Vessels sprouting from end of tied arteries.
18	Foà	—	Arch. per le Science Méd., iii, No. iv	Elements of repair derived from intima, and granulation tissue passing through the walls.
19	Froriep	—	Billroth, Lectures, &c., fig. 30, i, p. 157	Organisation of thrombus after ligature (drawing). Clot on distal and proximal ends.
20	Guthrie	1830	Diseases and Injuries of Arteries	—
21	Hamilton	1881	Edinb. Medical Journal, xxvii, p. 400.	Organisation of thrombus in ligatured artery chiefly due to penetration of vessels derived from vasa vasorum, pushing before them young connective-tissue corpuscles, which ultimately organise into fibrous tissue which replaces the thrombus, the clot acting simply as a support; a kind of sponge
	,,	1889	Text-book of Pathology, pp. 302, 307	
22	Hasse	1846	The Diseases of the Organs of Circulation and Respiration	—
23	Hogson	1815	Diseases and Injuries of Arteries	—
24	Holmes (T.)	1884	System of Surgery	Has always been an advocate of tight ligature on practical grounds.
			British Medical Association, Aug., 1890	

No.	Authors.	Date.	References.	Title or nature of contribution.
25	Hunter(John)	1785	Traité du Sang et des Inflammations (Trad. Richelot), t. iii	Vascularisation of clot beginning within the tissues of the vessel, as shown by injections. The question of organisation of the clot began to be brought forward by J. Hunter.
26	Jones (J. F. D.)	1805	A Treatise on the Process employed by Nature in suppressing Hæmorrhages, &c.	Inflammatory changes following ligation.
27	Lee	1870	Lectures on Subjects connected with Practical Pathology and Surgery, i	—
28	Lister	1881	The Lancet, i, pp. 201 and 275	On chromic catgut ligature; fibrous ring taking place of the ligature.
	„	1883	Lancet	Stagnation not sufficient to account for thrombosis.
29	Lobstein	—	Anat. Path., vol. i, p. 298	Axial vessel formed after ligature.
30	MacEwen	1878	British Medical Journal, ii, p. 359	Chromic catgut ligature proposed.
	„	1879	Id., i, p. 272	Young vessels sprouting from end of ligatured artery.
31	Manec	—	Traité de la Ligature des Artères	—
32	Moore	1870	Holmes' Syst. of Surgery, i, p. 712	—
33	Paget	1870	Lectures on Surgical Pathology, 3rd edit., pp. 160—208	References to Hunter, Stilling, and Billroth.
34	Pfitzer	—	Arch. f. Path. Anat., lxxvii, p. 397	Elements of repair derived both from the endothelium and granulation tissue passing through the walls of the vessel.
35	Pitres	1875	Archives de Physiologie	Recherches expérimentales sur le mode de formation des caillots.
36	Porta (L.)	1845	Delle alterazioni patologiche delle Arterie per la legatura e la torsione (Milano)	—
37	Quincke	1876	Ziemssen's Cyclopædia of Practice of Medicine, vol. vi, p. 363	Diseases of the Arteries. Reference to the views of Durante, Thiersch, Waldeyer, &c. Quincke does not believe that adhesions can take place between opposite walls of an artery so long as the intima exists.

No.	Authors.	Date.	References.	Title or nature of contribution.
38	Raab	1879	Langenbeck's Arch. f. klin. Chir., xxiii, p. 156	Ueber die Entwickelung der Narbe im Blutgefäss nach Unterbindung (full references).
	,,	—	Virch. Archiv, lxxv	—
	,,	1879	Centralbl. f. d. med. Wissensch., May 31st	Exclusive part taken by endothelium in organisation of thrombus.
39	Reckling-hausen	—	—	Thrombus takes no part in organisation. Organisation of thrombus through leucocytes.
40	Richardson	1858	Cause of the Coagulation of the Blood	Stagnation not sufficient.
41	Riedel	1875	Deutsch. Zeitschr. f. Chir., vi	Organisation of thrombus due to proliferation of endothelium. Adhesion of two endothelial surfaces may take place without intervention of a clot.
42	Rindfleisch	1872	A Manual of Pathological Histology, translated by Buchanan Baxter, i	Organisation of thrombus due to immigration of leucocytes. Absorption and softening of clot discussed.
	,,	1888	Traité d'Histologie Pathologique, traduit sur la 6me édit., allemande par F. Gross et J. Schmitt, p. 69	Id.
43	Rokitansky	1852	A Manual of Pathological Anatomy, iv, p. 327	Does not believe in vascularisation of the thrombus.
44	Schultz	1877	Deutsch. Zeitschr. f. Chir., ix, p. 84	Thrombus takes no part in organisation.
	,,	1878	Centralbl. f. d. med. Wissensch., ix, March 2	Leucocytes fed with cinnabar pass into the lumen of a vessel singly or doubly ligatured; intima takes no part. The clot is canalised from the lumen of the occluded vessel.
45	Senftleben (Cohnheim)	1879	Virchow's Archiv, lxxvii, p. 421	Senftleben experiment:—A large artery (aorta) of a rabbit doubly tied; the part thus cut off allowed to remain for three days, and then inserted into the peritoneal cavity of another rabbit, where the clot organises as in ordinary vessels.

No.	Authors.	Date.	References.	Title or nature of contribution.
	Senftleben (Cohnheim)	1880	Gazette Hebdomadaire de de Médecine et de Chirurgie, No. 20, p. 325	Supposed absence of clot when catgut used.
46	Shakespeare	1879	Toner Lectures, Smithsonian Institution, Philadelphia	Reparatory inflammation of arteries after ligature, acupressure and torsion.
47	Shakespeare and Simes	1880	Translation of a Manual of Pathological Histology, by Cornil and Ranvier	Difference in the mode of organisation of a clot not adherent and strongly adherent to arterial walls; when clot not adherent it is pushed up by the thickening of intima in the neighbourhood of ligature.
48	Stilling	1834	Die Bildung und Metamorphose des Blutpfropfes oder Thrombus	—
	„	1852	Rokitanski's Manual of Pathological Anatomy, iv, p. 327	Conical shape of clot, difference between proximal and distal ends. Injection of peripheral parts of young thrombus through main channel. In the old thrombi injection passes specially through the centre.
49	Syme	1848	Contribution to the Path. and Pract. of Surgery	—
50	Szuman	—	—	Formation of blood-vessels in clot.
51	Thiersch		Handbuch d. Chirurg., Pitha and Billroth, i, pp. 550—556	Organisation of thrombus beginning in the walls of the vessel, *i. e.* in the intima.
52	Tillmans	1879	Virch. Arch., lxxviii, p. 437	Experimentelle und Anatomische Untersuchungen über Wunden der Leber und Nieren.
	„	1881	Internat. Journ. Med. and Surg. N. Y., i, p. 223	—
	„	1881	Berl. klin. Wochensch., xviii, pp. 33, 35	—
53	Treves	1881	Brit. Med. Journal, i, p. 232	—
54	Vinay	1885	Dict. de Méd. et de Chirurg. pract., xxxviii, p. 786	Only remains of clot after organisation by growth of intima are a few hæmatic and other granulations.

No.	Authors.	Date.	References.	Title or nature of contribution.
55	Virchow	1856	Gesammelte Abhandlungen, Handb. d. Spec. Path.	Papers on blood coagulation, thrombosis, and embolism. Stagnation cause of thrombosis; great importance of clot in organisation.
56	Waldeyer	1867	Arch. für Path. Anat., xl, pp. 379—391	Zur pathol. Anatomie der Wundkrankh. Intima most important element in repair.
57	Warren	1886	Healing of Arteries after Ligature	—
58	Weber	1865	Handbuch der Allgemeinen und Speciallen Chirurgie, von Pitha und Billroth, Bd. i, Lief. 1, p. 143	Supposes that clot contains in itself the elements of organisation. Anastomosis of vessels formed in the clot with those of the tunica adventitia. Good diagram showing double origin of vessels of thrombus.
59	Zahn	1875	Virchow's Archiv, lxxii	Unters. über Thrombose.
60	Ziegler	1884	Pathological Anatomy, translated by Donald MacAlister, ii, p. 9	Organisation of thrombus, discussion of views. Extravasated leucocytes have the chief share in the formative process; the active participation of endothelial cells is not excluded. Good diagram of organisation of thrombus after alteration of constricted portion.

(For report of the discussion on this paper, see ' Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 153.)

SYMPTOMS AND PATHOLOGY

OF A CASE OF

ACUTE INFLAMMATION OF THE MUCOUS MEMBRANE OF THE ILEUM

FROM CLIMATIC CAUSES.

BY

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AND

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The case which is recorded in the following paper is that of a patient attended by Dr. Thin, who had known and seen him occasionally for a period of twenty years. A post-mortem examination was made after death, from an acute illness, by Dr. Martin, and the viscera were prepared by Dr. Wethered, and examined microscopically by Dr. Wethered and Dr. Thin.

Dr. Thin is responsible for the clinical history of the case. The pathological histology has been jointly studied by Dr. Thin and Dr. Wethered.

The patient died from a purely climatic disease, which

is certainly uncommon, looked at from the clinical aspect. Pathologically the authors have not been able to discover an account of exactly similar changes.

The patient was a strongly built, healthy, muscular man, æt. about 40, of excellent constitution and of remarkable bodily and mental vigour. He had spent about twenty years in different parts of China, during which time he visited England on several occasions, and during these visits it was observed that he enjoyed excellent health.

In October, 1888, when in the north of China, he began to suffer from occasional attacks of diarrhœa. He spent the winter travelling in more southern parts of China, the diarrhœa occurring at regular intervals all through the winter, although it was never severe. Returning to the north of China in the early spring of 1889, he received a severe chill, which was followed by fever. During March, April, and May the fever frequently recurred, the diarrhœa became worse, and in June both the fever and diarrhœa became persistent.

Medicinal and dietetic treatment failing to give any permanent benefit he went to Shanghai, where the diarrhœa abated under a diet of beef juice, but the fever remained. Having lost forty pounds in weight in four months he was ordered home, and during the voyage across the Pacific he lost five pounds more. In the meantime he had become very weak, and was sent to hospital at San Francisco in a state of great prostration, and still suffering from fever and diarrhœa.

After being forty-two days under treatment, twenty-one of which were spent in bed, he was able to continue his journey. He continued to improve rapidly, and after three months his strength was completely restored. He had gained thirty-six pounds in weight, and with the exception of an occasional loose morning stool considered himself quite well, and when we met him in April he seemed in perfect health.

The first symptom of indisposition occurred on the

15th of May, when diarrhœa, disturbed digestion, and loss of appetite followed a chill. Four days afterwards, finding these symptoms continue, he consulted Dr. Thin, and complained of abdominal malaise and irregular action of the bowels. He had no fever; the heart, kidneys, and spleen were healthy; there was no tenderness over the liver, the area of hepatic dulness being only very slightly in excess of normal. The tongue was of a dark red colour, cracked and furrowed, but quite free from fur.

He exposed himself freely to cold, caught a fresh chill two days afterwards, and after suffering very severe distress and fever for two days, during which time he took, at his own instance, castor oil twice, and several fifteen-grain doses of quinine, he sent for Dr. Thin, who found him in bed looking extremely distressed and ill, in profuse perspiration, with a temperature of 103°. He stated that he had suffered for two days from fever, profuse diarrhœa, and much retching. Another dose of quinine was administered, after which the fever disappeared for twenty-four hours. The following day, however, fever again set in, the temperature reaching 104°, with much nausea, and a continuance of profuse diarrhœa. After twenty-four hours' fever the temperature fell to 98° (on May 26th), and it remained normal for four days (until May 30th), with a slow full pulse, but during these four days the nausea, retching, diarrhœa, and general abdominal pain continued, the bowels acting four to five times daily. After these four days of apyrexia the temperature again rose to 103° on May 30th, and the febrile condition remained persistent till he died, three and a half weeks afterwards, on the 25th of June.

We believe the chief features of the case will be more readily understood if we deal with the most important symptoms separately, instead of describing the condition from one day to another.

1. *The fever.*—The chill which he received on the 21st of May was followed by a severe rigor and fever. After two days of a temperature varying from 102° to

103° the temperature remained normal for twenty-four hours, when it rose again as high as 104°, but again subsided to normal after a day's fever, and the patient remained for four days with a normal temperature. Then on the ninth day of illness the temperature rose to 103°, and he had a febrile temperature from that day until he died, twenty-five days afterwards. There was a daily rise, which usually took place at night, sometimes slightly exceeding and sometimes not quite reaching 103°, the daily minimum being usually a little over 101°. It fell slightly on June 20th, when he began to pass blood from the bowels, and rose again the day before his death.

2. *Nausea and retching.*—From the first day of the attack, on the 21st May, until the day before his death, nausea was more or less persistent, not being much felt for hours at a time if nothing was swallowed, but always evoked to some extent, even by swallowing fluids. Retching was a prominent symptom during the whole illness, and could only be kept in check by very small quantities of fluid being swallowed at a time. Milk was nearly always vomited, even when taken in the smallest quantities, and even thin water arrowroot was not borne. The only nourishment that the stomach could receive was very small quantities of the juice of raw meat, or one or two teaspoonfuls of concentrated chicken broth. Any attempt to assuage the incessant thirst by drinking fluids usually brought on severe vomiting.

3. *Diarrhœa.*—Diarrhœa began with the sudden onset of the illness on the 21st of May, and continued until the end. During the first two days the diarrhœa was very frequent, but afterwards the motions were usually four to six daily. They were passed without pain or straining. The character of the stools remained the same from the beginning until within a few days of his death, when they contained fluid and coagulated blood. They were quite fluid, not very copious, of a dark brown colour, somewhat resembling dark mahogany. This colour never varied

throughout the illness. They had a sickening though not very intense odour, but the smell was not fæcal. They contained nothing resembling fæces from beginning to end. On June 20th, five days before his death, dark blood-clots were observed in the motions. These increased in quantity until the amount of blood lost by the bowel was considerable, and this bleeding undoubtedly hurried on the catastrophe.

During the first days of the illness he complained of some abdominal pain, but the tenderness was not great: in its subsequent course there was at times a sensation of uneasiness, scarcely approaching to pain, felt more or less during the whole time, but on some days it was scarcely noticed, and pain was certainly not a prominent symptom. On firm pressure over the bowels, particularly in the umbilical region, some pain or uneasiness was evoked, but the tenderness to pressure was not marked. On very firm and hard pressure on the lower edge of the liver slight pain or uneasiness was also produced, but this again was not a marked symptom.

The appearance of the tongue varied very little from the first, remaining of a dark red, and free from fur.

His mind continued clear and unclouded until within a few days of his death. Simultaneously with the hæmorrhage from the bowels he had short intervals of slight wandering, scarcely approaching delirium, followed by intervals of clearness.

He closely observed his symptoms, and reasoned from them very clearly.

In the third week of the illness he came to the conclusion that recovery was very improbable, and shortly afterwards gave himself up as being hopelessly ill, being much impressed by the continued febrile condition, and incapacity to take or retain food.

During the whole course of the malady there was not only an entire absence of appetite, but the foods usually given to invalids were almost invariably rejected. Digestion, as we shall immediately see, seemed to be entirely

suspended. Emaciation, which was not rapid, progressed steadily until the end. The pulse was, considering the severe nature of the symptoms, never very quick, the patient naturally having a slow pulse. It was only the day before his death that it rose above 100, when it rapidly increased till it reached 140 about ten hours before his death.

In this account of the illness we have purposely omitted any reference to the treatment by drugs and by diet, in order that the clinical picture might be less disturbed; but before remarking on these two points it will be convenient to record what the patient told us concerning his acute illness in San Francisco.

The attack that he had there was evidently of the same nature as that from which he eventually died, the chief symptoms being retching, passage of dark-coloured fluid motions by the bowels, abdominal uneasiness, and fever. It was found at San Francisco that milk could not be borne, the food on which he eventually recovered there being pure beef juice squeezed from raw meat, of which he took from five to six coffee-cupfuls a day. During his last illness it was also found that milk could not be borne, producing invariably more or less sickness, and that which was not vomited appeared in the stools as white cheesy coagula. Yolk of egg was tried, and passed undigested in the motions. Raw white of egg in water was rejected. Thin barley water was given him for his thirst, water arrowroot was also given when the temperature was at its lowest. Raw beef juice was freely and persistently tried; he had also freshly prepared beef and chicken jelly, and occasionally whey. These were given in the very small quantities which the stomach tolerated, and seemed to produce no effect on the colour or consistence of the intestinal discharges.

During the first week of his illness quinine had a decided and marked effect in reducing the temperature. It was continued, usually in full doses, until the last week of his illness, but latterly with less effect. No quinine

being given from the 19th to the 22nd, it was found that the temperature was on an average one degree higher; and on being again administered, the temperature immediately fell to what it was before. The distressing nature of the symptoms appearing to depend largely on the fever, the quinine was usually given in doses of five grains, as much as twenty grains being sometimes given daily.

When it was found that full doses of quinine failed to reduce the temperature, four grains of antifebrin at once lowered it and produced great relief; but the depressing effect of this drug was so marked that it was discontinued. As soon as the symptoms of hæmorrhage from the bowels appeared ergot was given, usually in the form of hypodermic injections of ergotin, but without producing any apparent results.

Occasionally moderate doses of laudanum were tried, without producing any effect on the diarrhœa. During the last week of his illness, when the distress became more aggravated, hypodermic injections of morphia were administered, without, however, producing very happy effects. Death occurred from exhaustion, accompanied by distressing nervous symptoms, on the 25th.

The general points to be noted in the case appear to be that for about a period of thirty-three days there was an apparent almost entire, if not entire, suspension of the digestive functions; the absence of anything like fæcal matter in the fluid intestinal discharges; and persistent fever (with an intermission of low temperature, lasting for four days) after the first three days of his illness. The tendency to nausea and occasional retching persisted the whole time. Abdominal uneasiness, although never severe, was hardly ever absent.

On account of Dr. Wethered's absence from town the post-mortem examination was made by Dr. Sidney Martin, whose report is as follows:

Æt. 41. Date of death June 25th, 1890. Date of examination, June 26th, 1890. Weather mild, wet. Rigidity slight.

General appearance.—Body emaciated but not extremely. Very little subcutaneous fat. Muscles slightly developed. No œdema.

Heart.—Pericardium, no excess of fluid. No pericarditis. Valves and endocardium normal. Muscles pale, otherwise normal.

Lungs.—Pleuræ not adherent; no excess of fluid. Lungs slightly congested posteriorly, otherwise normal.

Abdomen.—No ascites or peritonitis.

Liver.—Slightly enlarged; substance pale and rather soft (slightly fatty). On upper surface in the substance of the right lobe is a wedge-shaped mass about the size of a large bean; pale red on section, firm; it has more the appearance of a vascular growth than of an infarct.

Spleen.—Enlarged slightly, soft, diffluent. Near surface of spleen are three masses similar to that found in liver, one quite round; size of a large pea to a small horse-chestnut; largest is wedge-shaped. Masses are dark red, firm, not caseating; somewhat resemble recent infarcts.

Kidneys.—Not enlarged; capsule not adherent, surface smooth. Cortex not diminished. Substance pale, firm (anæmic).

Stomach.—Contains mucus and bile-stained food; no blood. Mucous membrane normal.

Small intestine.—The mucous membrane of the jejunum is bile-stained (green); in the upper part it is for the most part normal, showing two or three superficial erosions size of a split pea. The mucous membrane of the lower part of the jejunum is pigmented in dots, giving the "shaved beard" appearance (chronic inflammation).

The mucous membrane of the ileum and of the large gut is covered with dark brownish-red and liquid blood, which stained the surface of the gut. On washing the blood away the mucous membrane presents a worm-eaten appearance along the whole course of the ileum to the cæcum. This sinuous ulceration does not go deeper than the mucous membrane; in some places there are discrete

erosions size of a split pea. The ulceration increases as it descends the ileum. The mucous membrane is thickened generally, but the other coats of the gut are not affected.

Large intestine.—Just at the entrance of the vermiform appendix the mucous membrane of the cæcum is thickened and slightly eroded. There are about ten superficial ulcers in the large intestine; five are grouped together. None is larger than half an inch long and a quarter of an inch broad. Their edges are sharp-cut, not thickened, and the base formed by the lower layers of the mucous membrane and by the subcutaneous coat. The mucous membrane is not generally thickened; one or two of the ulcers appear to be in the solitary glands.

Mesenteric glands.—Not enlarged.

The specimens were placed for a few hours in alcohol, and then transferred to a 2 per cent. solution of bichromate of potash. After an interval of about six weeks they were embedded in celloidin, and the sections stained in hæmatoxylin and eosin.

Microscopical examination of the tissues thus prepared showed that there was nothing in the condition of the liver, spleen, stomach, jejunum, or heart of a markedly abnormal character. The pale wedge-shaped masses described by Dr. Martin in the liver and spleen were found on microscopical examination to consist of localised dense small cell infiltration around the vessels. We regard them as the first stage of a congestion that would have led to the development of pyæmic abscesses had the patient lived longer. The mucous membrane of the large intestine was found to be perfectly healthy. This remark of course applies to the gut as a whole, and not to the few superficial ulcers close to the cæcum.

The histological changes, as well as the naked-eye appearance, showed that the disease was essentially, and it may be said almost exclusively, an acute process in the mucous membrane of the ileum.

The changes in the ileum were found in the following stages.

No traces of the villi were seen in any of the sections, the free surface being composed of a seam, more or less deep, of a granular material, which was coloured a uniform brick-dust colour by eosin, and in which no cellular elements were stained.

At the parts least affected, Lieberkühn's crypts seemed in the deeper parts to be unchanged. Towards their extremities the epithelial cells were difficult to make out, being rebellious to the action of the staining fluids used; but they did not seem broken up. Towards the free surface they became lost in the layer of finely granular material which covers the surface of the mucous membrane.

In the next stage observed the crypts are shorter than usual; the lumen distended with a clear somewhat glutinous-looking substance in which fine granules are scattered. The epithelial cells are more or less broken up and vacuoled. The shortened crypts soon end in a thicker and better marked layer of the finely granular material previously referred to. This condition may be described as an atrophic condition of the epithelial structures involving a certain amount of shrinking and destruction.

A more advanced stage was best seen in the intermediate parts between the slightly affected crypts and the eroded or superficially ulcerated parts. In this stage the outlines of the crypts could still be traced by the rows of disintegrated and partially destroyed epithelial cells. The nuclei of the epithelium in this stage refused to take on any stain, and the crypts were in an advanced stage of destruction and disintegration, being lost in the surrounding and covering granular layer previously alluded to.

The last stage is that of erosion, or superficial ulceration. All traces of epithelium or crypts are gone, the free surface being composed of a thick layer of the granular material referred to. In the deeper parts of this layer leucocytes commenced to be made manifest by the action

of dyes, and in the deepest parts of it they were present in dense groups.

When the whole thickness of the ileum was examined, it was found that small-cell infiltration was present unequally in the submucous layer, very dense in the parts which were eroded, and partially and unequally in the parts which were less affected. Nothing remarkable was observed in the subperitoneal layer or the muscular coats. Under the eroded parts a certain amount of cell infiltration was present around some of the vessels in the inner muscular coat.

In some sections in the subserous layer, which was not specially congested, or the seat of any infiltration, large ovoid cells were seen, with granular protoplasm well stained, exactly similar to cells frequently seen in tissue undergoing repair, or in rapidly developed soft fibrous tissue in certain pathological conditions. These were possibly indicative of reparative changes that had followed the patient's recovery from the acute illness which he had in San Francisco.

In comparing the clinical symptoms with the naked-eye and microscopical pathological appearances, we believe we are justified in coming to the conclusion that we have had to do with an acute inflammation of the mucous membrance of the ileum supervening on imperfect recovery from a previously acute attack.

From the history given by the patient it seems clear that the first appearance of the disease was insidious and not attended by fever. After lasting for some months the process became subacute, and was attended by fever. On the subacute condition there developed a highly acute process, from which complete recovery took place, leaving, however, a susceptibility which, under the influence of accidental causes, led to the development of a second very acute and fatal attack.

The unknown cause of the disease which led to the death of this strong man was one that found the conditions of its development and activity in the mucous mem-

brane of the ileum, and did not find a congenial soil in any other part of the intestine; because, considering the healthy condition of almost the whole of the large bowel, we can only regard the limited affection of that gut near the cæcum as a very partial continuation of the disease dependent on the condition of the ileum.

A case previously published by one of us (see 'Brit. Med. Journ.,' June 14th, 1890) establishes the fact that in a peculiar, insidious, chronic climatic disease, unattended by fever, psilosis of the bowel, or "sprue," the mucous membrane of the ileum is the special seat of election of a morbid element; but these cases are conspicuous by the entire absence of fever.

It is possible, in the case we are now discussing, that from exceptional causes a similar affection of the ileum became, and continued, more or less acute; but if we were to look at it in this light, namely, that of acute congestion of the mucous membrane of the ileum, supervening on the early stage of psilosis, the case would require to be regarded as very exceptional. Favouring the idea that it was an exceptional acute condition of the usually chronic disease is the fact that anatomically the same portion of the gut was exclusively affected, and that in the early stages of the malady, before it had become acute, the patient—a highly intelligent man—identified his case as being similar to those described by one of us under the name of psilosis. In this case, however, there is no record of the peculiar condition of the mucous membrane of the mouth and gullet which is characteristic of the chronic wasting process.

Clinically it is to be observed that in this case the general symptoms of acute congestion of the mucous membrane of the ileum were retching, nausea, complete anorexia, and great thirst, the temperature ranging from 101° to 103°; the discharge from the bowels of a dark-coloured fluid, in which there was no apparent trace of fæcal matter, absolute suspension of digestion, and steadily progressive emaciation.

The diseases from which it was necessary to distinguish it, and in regard to which a question of diagnosis naturally arose, were typhoid fever and a particular form of dysentery. From typhoid fever it was distinguished by the absence of spots and of tympanites, by the unclouded intellect, which remained perfectly clear until almost the end, when the patient was under the influence of morphia, and by the entire absence of the characteristic typhoid stools.

There is a form of dysentery in which, from the rapid destruction of the mucous membrane of the large intestine, almost from the beginning of the disease considerable quantities of a highly offensive dark-coloured fluid are poured from the bowel. The fluid of these dysenteric cases has somewhat of the tint of Condy's fluid, and is not so brown as the discharges were in the present case. Exhaustion rapidly takes place, and the patient dies long before the term reached in the case which we have described.

In the present case, the knowledge that the mucous membrane of the ileum was liable to be specially affected, the previous history of the case as given by the patient, and the exclusion of all other abdominal diseases known to us, led one of us to make the diagnosis of acute congestion of the mucous membrane of the ileum at the very outset of the illness, and he never saw any reason to depart from his view during its whole course—a view which, as we have seen, was completely borne out by the post-mortem examination.

In regard to the complete suppression of the digestive functions, whilst the whole of the intestinal viscera except the ileum were comparatively healthy, we would suggest that it is a question as to how far the functions of the anatomically unaltered digestive glands were suspended by a reflex effect produced by the congested mucous membrane of the ileum.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 165.)

DESCRIPTION OF PLATE VII.

Symptoms and Pathology of a Case of Acute Inflammation of the Mucous Membrane of the Ileum from Climatic Causes (GEORGE THIN, M.D., and F. J. WETHERED, M.D., M.R.C.P.).

FIG. 1.—*a*. Submucous layer.
b. Lieberkühn's crypt, very slightly obliterated at the free surface.
c. Remains of a Lieberkühn's crypt, destruction almost complete.
d. Dense small-cell infiltration of submucous layer under superficial ulceration.

FIG. 2.—Oblique section of a Lieberkühn's crypt showing breaking up of some of the epithelial cells. The cavity of the crypt is filled with a transparent glutinous-looking substance with granules.
a. Membrana propria of the crypt.

FIG. 3.—Section through one of the small ulcers showing the subperitoneal and muscular coats unaltered.
a. Subperitoneal coat.
b. Outer muscular layer.
c. Inner muscular layer, slight small-cell infiltration round some of the vessels.
d. Dense small-cell infiltration in the submucous layer.
e. Granular substance covering the free surface of the ulcer.

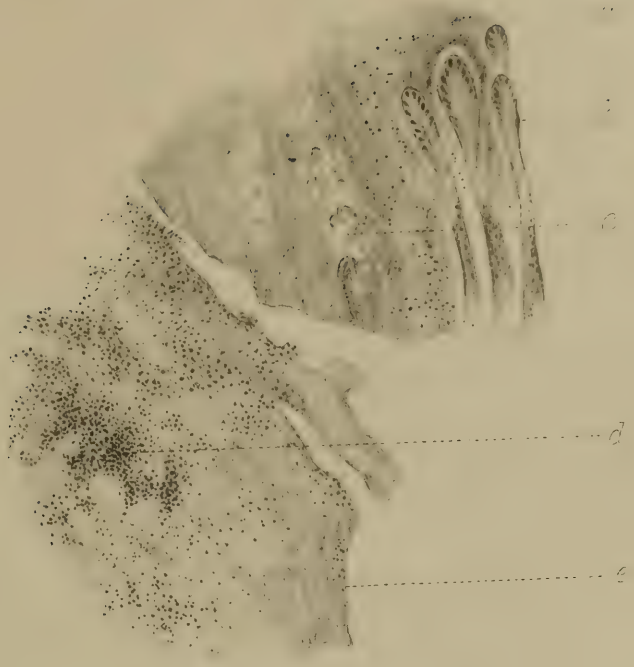


Fig. 1.

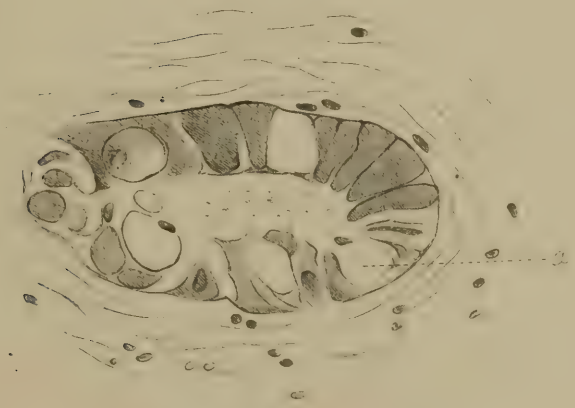


Fig. 2.

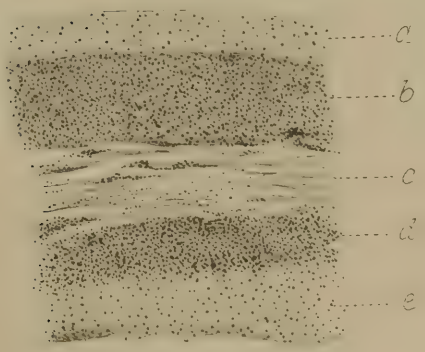


Fig. 3.

A. Danielsson, del.

A CASE OF PANCREATIC CYST

SUCCESSFULLY TREATED BY ABDOMINAL SECTION AND DRAINAGE.

BY

G. NEWTON PITT, M.D.,

AND

W. H. A. JACOBSON, M.Ch.

Received March 10th—Read June 9th, 1891.

Benjamin McG—, æt. 21, a slater, was admitted into Guy's Hospital under Dr. Perry with jaundice and epigastric pain on May 29th, 1889. A sister in the previous year had had an attack of jaundice, which had lasted for a short time, for which no very obvious cause was found. He has been a free drinker of beer and spirits.

Three years ago he received a kick in the abdomen, which caused him severe pain and sickness, confining him to bed for three weeks. There was no external bruising, no hæmatemesis, and no melæna. Ever since the accident he has been liable to attacks of pain, which last from a few hours to a week. The attacks have no relation to food, they are often more severe in the night, and at times are accompanied with sickness.

In 1888 he had an attack of jaundice, which lasted for two or three days. Three weeks previous to his admission he became jaundiced, and its intensity has increased

gradually. His skin has itched severely; his abdominal pain at the present time is not more severe than usual. He has been sick on several occasions, and his appetite has been bad.

Condition on admission.—A sparely built man, with very thin limbs, which condition he says is of recent date. He is markedly jaundiced; the skin is covered with scratch marks. There are no evidences of syphilis; his fingers are very slightly clubbed.

His bowels are constipated, his appetite is bad, there is a constant feeling of nausea, and when the pain is severe, he is sick.

The abdomen moves normally; the muscles in the epigastric region are rigid. On percussion, there is dulness in the middle line, from the xiphoid cartilage to half an inch below the umbilicus; on the left it extends rather lower down, and outwards as far as the border of the rectus. Towards the right the area of dulness curves upwards to the margin of the ribs. In the left portion of the epigastric and umbilical regions an elastic, ovoid mass can be felt, with a well-defined edge, moving with the liver in respiration. It is tender, and there is a continuous pain. No fluctuation can be made out.

He has also attacks of pain of a much more severe character, always localised in the same area, of a dull aching character, usually worse at night. The pain has none of the characters of hepatic colic, never radiating to other parts; it is never sufficiently violent to double him up. He has had no rigors; the spleen is not palpable. The respiratory, circulatory, and nervous systems are normal. The urine is of sp. gr. 1022, acid in reaction, dark brown in colour. It contains bile-pigment, but is free from albumen and sugar.

June 3rd.—The amount of pain has been very variable, but was more severe last night. The note on percussion has quite altered to-day. Over the swelling, and towards the right, the note is hollow and of a boxy character, though duller here than in the epigastrium. On bobbing the finger

down suddenly on to the abdominal swelling, air and fluid are displaced with a sudden gurgle from over the swelling, and less markedly from over the right side. The edge of the liver is still palpable. The patient took iodide of potassium, with hyoscyamus at night when necessary, and belladonna was applied locally.

FIG. 1.

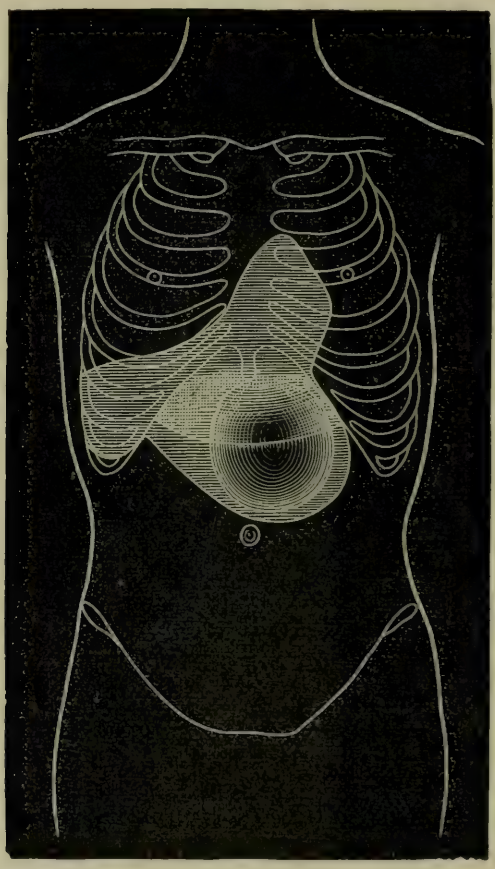
The area which, early in June, 1889, was dull on percussion, is shaded with horizontal lines. The projecting mass of the tumour is also indicated.

13th.—Mr. Symonds aspirated the swelling this afternoon, drawing off ten ounces of opaque greenish fluid, alkaline in reaction, sp. gr. 1015, containing abundant albumen, but no bile. Under the microscope red blood-cells and a few pus-cells were visible, but no echinococcus

hooklets. The swelling was much reduced in size. After the tapping the patient had increased pain, which was relieved by a morphia injection.

14th.—The patient is much freer from pain this morning, the jaundice is less marked, and the lower limit of the dulness has receded two inches.

FIG. 2.



The area which is shaded with horizontal lines was dull on percussion in August, 1889. The prominent portion of the tumour is also indicated.

16th.—The pain has again increased considerably, and the region of the swelling is very tender. The pain together with the jaundice and the swelling gradually diminished, so that on the 26th the patient was able to go home, there being only a slight amount of jaundice.

At the end of July the pain returned; it was continuous and increased in severity. The patient was readmitted under Dr. Newton Pitt on August 12th, it having been noticed that the swelling had returned a week previously. He was still jaundiced.

Condition on readmission.—There is a deep-seated tumour in the upper part of the abdomen, which pushes forward the abdominal walls in the epigastric and umbilical regions from one to one and a half inches, extending further to the left than to the right of the middle line. The left flank is the fuller; the umbilicus is depressed. On palpation the swelling is found to extend for some distance transversely, especially to the left.

The surface is convex and uniform, except for an ill-defined transverse rounded ridge across its most prominent part. The rounded swelling can be felt reaching from below the tip of the ensiform cartilage to just above the umbilicus, and laterally to near the tip of the eleventh rib, especially on the left side. The right rectus is more tense than the left, but the abdominal walls are rather flaccid. The tumour moves down freely on inspiration, otherwise it possesses little or no mobility, and gives the impression of being attached to some deep-seated structure. There is a transmitted impulse synchronous with the pulse, but the impulse is not expansile. The tumour feels hard and firm, no fluctuation can be made out. On pressure just below the ensiform cartilage, gurgling can be made out. No other abdominal tumour can be felt.

Measurements.—The tumour measures vertically five inches, and transversely seven and a half.

	Inches.
From the umbilicus to the ensiform cartilage is . . .	5¼
„ „ „ pubes	5¾
„ right anterior superior spine to the ensiform cartilage	10
„ left „ „ „	9½
The left half-circumference from the umbilicus to the spine is	13
The right „ „ „	13
The left half-circumference from ensiform cartilage to the spine is	14¼
The right „ „ „	15⅛

On percussion there is dulness over the most prominent part of the tumour, and around this an area of tympanitic resonance. To the right the dulness merges into that of the liver; on the left the stomach resonance is normal, except near the middle line, where it is encroached upon by the tumour dulness, the tympanitic note here not being quite so clear as normally. There is tympanitic resonance between the tumour and the margin of the ribs on the left side. The resonance over the lower part of the abdomen is normal, except for a slight deficiency in the left flank. The liver dulness is normal, and there is no evidence that the margin extends beyond the ribs.

The splenic dulness is normal. No hydatid thrill can be made out.

The bowels are constipated. The urine is of a sp. gr. of 1015, acid, free from deposit, sugar, and albumen. It is dark-coloured from the presence of bile-pigment, and contains 3 per cent. of urea.

August 14th.—The physical signs have changed considerably to-day. On palpation the tumour was found to be less superficial, and could not be traced over as large an area as that of yesterday. To-day in front of the tumour there is a sac containing fluid, which by bobbing suddenly can be made to gurgle and splash. On light percussion there was tympanitic resonance over the surface of the tumour, with an occasional succussion sound. The stomach resonance extended from its normal confines over the surface of the tumour, reaching nearly to the right limit of the epigastrium.

Again to-day the same changes in the physical signs were observed. Before food the tumour dulness was extensive and superficial; after food there was every evidence that the stomach lay between the tumour and the abdominal wall.

16th.—When the stomach was empty, and the tumour gave a dull note on percussion, a seidlitz powder was administered in two parts in order to distend the viscus with gas. Succussion could now be readily obtained over

the tumour area, and the note on percussion was tympanitic. It was, therefore, clearly proved that the stomach lay in front of the tumour.

17th.—The patient was kept without food in the morning, and in the afternoon a trocar and canula (one sixteenth of an inch in diameter) were passed into the tumour near the seat of the old puncture, and eight ounces of fluid drawn off. The canula was withdrawn before the fluid had quite ceased to flow.

The fluid was alkaline, sage-green in colour, sp. gr. 1013, containing 11 grams per litre of albumen. It contained no copper-reducing substance, and fuming nitric acid gave no bile reaction. When the fluid had settled for twelve hours, numerous small white particles had gravitated to the bottom, but the fluid still remained turbid.

Under the microscope the turbidity was found to be due to innumerable collections of globular masses and of scattered crystals. The crystals were colourless, acicular, and somewhat curved. Most of them were aggregated into globular masses, but some were arranged in bundles.

When treated with H_2SO_4 and Fe_2Cl_6 the purple reaction of tyrosin was obtained. The crystals were those of tyrosin; no leucin could be detected either by chemical reaction or by the microscope. When the fluid had been allowed to stand in a warm place for twenty-four hours with some starch, it contained a copper-reducing substance. On decomposition the fluid gave off abundance of H_2S, but even after four days the red blood-cells retained their form. At this time decomposition bacteria abounded. From the position of the stomach as mapped out by percussion during different stages of distension it was clear that the trocar had passed through both its walls to reach the tumour; this was also corroborated by its site at the time of the operation. No food was given by the mouth for twenty-four hours, but the patient was fed by nutrient enemata. During the succeeding twenty-four hours the pain was very severe, necessitating subcutaneous injections of morphia.

19th.—The tumour has partially refilled, and is tense.

21st.—The tumour is as large as on admission.

Mr. Jacobson saw the patient in consultation with Dr. Pitt, and it was agreed that the tumour was a pancreatic cyst, which was rapidly becoming so tense and painful that it was desirable to drain it as soon as possible. The necessity for the operation and its dangers having been placed before the patient, he was desirous that it should be performed.

These cysts have several times been correctly diagnosed, either from their site or from the character of the fluid obtained by aspiration. In this case, the evidence that the tumour was a pancreatic cyst appeared to be conclusive. The nature of the fluid contents (amylolytic and containing tyrosin) and the symptoms agree with those which have been met with in undoubted pancreatic cysts, and the tumour was situated behind the stomach in the region of the pancreas.

Further evidence appeared unnecessary, and could only have been furnished by a post-mortem examination.

22nd.—The patient has steadily emaciated since his admission, although even then he was very thin. He has more or less constant severe abdominal pain, with frequent exacerbations. He is jaundiced, and is most seriously ill; there can be no doubt that surgical interference offers him the only chance of recovery. The tumour is more prominent, and occupies a larger area than on admission. Over the tumour there is complete dulness, which extends more to the right than to the left, and is continuous with that of the liver. At 2 p.m. Mr. Jacobson opened the abdomen, using the carbolic spray. A longitudinal incision three inches long was made one and a half inches to the left of the middle line, extending to within an inch of the umbilicus. The parietal layer of the peritoneum was divided and attached to the skin round the cut edge, by means of sutures at intervals of one third to half an inch, care being taken that no muscle intervened between the two surfaces that it was desired to unite. This was rather difficult to accomplish, as there was great tension owing to the size and

bulging forward of the cyst. At the upper part of the opening the lower edge of the liver could be seen moving with the respiration. Below this, covering the tumour, was a smooth, reddish, vascular surface, which bulged strongly forwards.

Taking this to be the part of the cyst which had been ascertained before the operation to be dull on percussion, Mr. Jacobson intended to leave this for twenty-four hours, to become adherent before it was incised. The result showed that if this had been done the scalpel would have passed through the walls of the stomach. The surface of the cyst being again scrutinised Mr. Jacobson thought that he found evidence of involuntary muscular fibre, which threw doubt upon the swelling being pancreatic. The wall of the supposed cyst being examined between the fingers, proved to be the empty stomach stretched tightly and smoothly over the subjacent cyst. To explore this the stomach was drawn upwards, so that it might be packed away above under the liver. But then arose an embarrassing difficulty. As the stomach, which was tightly jammed between the bulging cyst behind and the abdominal wall in front, was raised, the omentum came up into the wound in front of the cyst. The tension of the parts was so great, owing to the rapid increase in the cyst, that there was no room above in which to pack away the omentum. Pushing this to either side, regions already fully occupied, pulled the stomach down again over the cyst. The greater part of the omentum was accordingly drawn out of the wound. Some of it was tied with gut and cut away, but much of it was left heaped up on the abdominal wall on either side of the incision. One or two catgut sutures retained this in position. The two layers of omentum which (just below the stomach) still lay in front of the cyst were next scraped through, exposing the surface of the cyst for a space the size of a shilling. This was very vascular, and so extremely tense that it was not thought advisable to put in a guiding suture. Thus there was a somewhat conical passage leading from the abdominal incision, through a mass of omentum,

down to the anterior surface of the cyst. Iodoform having been dusted on, dry sal-alembroth dressings were applied.

It is easy to see now, after the event, that with so large a cyst, and one that made tension and displacing pressure upon its very important surroundings, it would have been better surgery to have emptied the cyst at once, either by means of a large trocar and tubing, or by opening the cyst freely, seizing the cut edges with Spencer Wells' forceps, and thus holding the cyst well forwards, while the patient was turned on his side. The cyst would then have been sutured to the parietal incision in the usual way. This course would have prevented the very grave risk which befell the patient a little later.

23rd.—The patient was fed every three hours with nutrient enemata, and only a very little iced water was allowed by the mouth. At midnight he was very collapsed and in great pain, in spite of a subcutaneous injection, at 6 p.m., of a third of a grain of morphia. His pulse was 130, and was feeble. The injection of morphia was repeated, but his pulse became more rapid, reaching 163 at 2 a.m. At 3 a.m. his condition was so precarious that Mr. Jacobson let out with a hydrocele canula twelve ounces of fluid, which had been pent up under such high tension in the cyst as to spring forwards for several feet. The rest of the fluid was removed through a large canula, the sac was incised, and a large drainage-tube was inserted. The adhesions of the omentum to the abdominal wall were but slight. Sal-alembroth dressings were reapplied. The urine contained albumen and a trace of sugar.

24th.—The urine is acid, sp. gr. 1027 ; it contains neither casts nor sugar, and only a trace of albumen. The fluid evacuated from the sac is dark port wine in colour, sp. gr. 1029, and gives a spectrum of methæmoglobin. The fluid becomes solid on boiling. Under the microscope there are leucocytes, red blood-cells, and large epithelioid cells, with many fat granules visible. The patient's condition is better to-day, his pulse is 160 ; his temperature in the

morning was 100°, but it became normal in the evening. In addition to the rectal nourishment, he has taken by the mouth two drachms of brandy and half an ounce of champagne alternately every half-hour. He has had morphia both subcutaneously and in a suppository.

25th.—During the past twenty-four hours he has improved considerably, his pulse has become stronger, the rate has decreased to 116, and the jaundice is less marked. The morning temperature reached 100°, and he still feels depressed. He has taken by the mouth brandy, port wine, and half a pint of milk.

26th.—From this time he steadily improved, the jaundice gradually disappeared, the temperature remained normal, and he was almost free from pain.

29th.—The abdomen is dressed twice daily, the discharge consists of about a drachm of dark treacly fluid. The urine is normal.

September 4th.—The irritation set up by the discharge has produced a vesicular eruption round the incision.

12th.—The discharge has become serous.

20th.—The drainage-tube was removed, leaving a sinus one and a quarter inches long. He has a pain in the hypochondrium after drinking.

22nd.—He was up for the first time; the sinus is practically closed, only a few drops of fluid exuding. The omentum had by this time shrunk and shrivelled away, having merged in the granulations round the wound. He went home shortly afterwards with the sinus completely closed. He felt well and had regained flesh.

There are several features of interest in this case.

In the first place it is the only instance in Great Britain, with the exception of Prof. Annandale's case, in which a pancreatic cyst has been correctly diagnosed previous to drainage.

The formation of the cyst was attributed to a kick in the abdomen, which the patient had received three years previous to admission, since which time he has been liable to attacks of pain. When admitted, he was emaciated and

jaundiced, and there was also a history of a temporary attack of jaundice in 1888.

Among the recorded cases, jaundice was noticed three times, pain eight times, emaciation five times, a history of injury three times.

The nature of the fluid which was removed in June was similar to that usually found in pancreatic cysts; its alkaline, turbid, albuminous nature excluded the possibility of its origin from a hydatid, an omental, or a supra-renal cyst, or from a localised peritoneal collection.

When the patient was readmitted in August, 1889, it was clear that there was a globular tense cyst lying behind the stomach, and the fluid which was obtained from the second tapping was so characteristic that there was no doubt as to the diagnosis.

This fluid was alkaline, sage-green in colour, sp. gr. 1013, and turbid. It contained 1·1 per cent. of albumen, also tyrosin crystals in abundance, and a few pus-cells. It was free from sugar and bile-pigment, but contained a sugar ferment.

The intermittent character of the jaundice indicates that the cyst arose from the central portion of the pancreas, and when the cyst became tense it caused the jaundice by pressing upon the head of the pancreas and the common duct.

One of the most noteworthy features in the case was the almost fatal condition of collapse and cardiac failure which ensued thirty hours after the cyst was attached to the abdominal wall. This fixation necessarily caused some traction upon the solar and other nerve plexuses which lay behind the tumour, and the consequent nerve disturbance produced great cardiac depression. Within a few hours the patient was profoundly collapsed, with a very feeble pulse, over 160 per minute, and suffering intense abdominal pain. It was only by tapping the extremely distended cyst thirty-six hours after the operation that a fatal termination was warded off, and for many hours the patient's life hung in the balance.

Aspiration may be desirable in these cases for the purpose of diagnosis, but there is no evidence that it can ever be curative. With care there is not a great risk, but there is always the possibility, as has already happened in some cases, that the puncture may continue to ooze, and necessitate laparotomy at once.

Attempts to remove the cysts completely have with two exceptions proved fatal; while abdominal section, with drainage of the cyst, has been successful in nearly 90 per cent.

It is clear, therefore, that incision and drainage of the cyst is the treatment to be recommended.

We venture to submit to the Society the following points for discussion :

1. The mode of origin of cysts of the pancreas.
2. The diagnosis of these cysts, which since the publication of Professor Senn's papers have been proved to be far less uncommon than was previously thought.
3. The liability of hæmorrhage to take place into them, causing very grave and urgent symptoms as in our case.
4. The question of the advisability of puncturing them from the front.
5. The best site for opening and draining these cysts.

1. *Mode of Origin of Pancreatic Cysts.*

Injury has been a prominent feature in the history far too frequently to be neglected. Thus this was clear and marked in the cases of Kulenkampf, Fenger, Senn, Karewsky (two cases), Riegner, Treiberg, Cathcart, and our own. There was also an injury, but less clearly connected with the formation of the cyst, in the cases of Steele and Küster. The injury, even in so well protected a viscus as the pancreas, probably produces some laceration and extravasation. With the latter is mixed the constantly increasing fluid from a torn duct. As pointed out by Cathcart (l. c.), this collection of fluid probably becomes irritating, and thus excites the formation of a capsule about it.

Of the facility with which hæmorrhage may occur into such a cyst we have spoken later. Other theories, ingenious but as yet wanting in proof, have been put forward by Gussenbauer and Salzer. According to them these cysts originate in some digestive or corrosive action of the pancreatic juice upon the tissue of a previously diseased pancreas. A cystic pouch is thus formed, into which hæmorrhage may easily take place, either from a vessel on the wall of the cyst, or from one lying in an intra-cystic partition which has given way.

Finally Dr. Boeckel, of Strasbourg ('Des Kystes pancréatiques,' Paris, 1891), suggests that the relations of the pancreas, especially its head, with the duodenum, establishes a direct connection between the inflammations of the intestines and the origin of pancreatic cysts, the inflammation being propagated from the intestine to the gland by the pancreatic duct. Inflammation of the duct wall brings about its contraction, and thus an accumulation of fluid, and finally the formation of a cyst.

2. *The Diagnosis of Pancreatic Cysts.*

Since attention was directed to these cysts by Senn, in the 'International Journal of the Medical Sciences' in 1886 and 1887, when he grouped together the main symptoms and physical signs, correct diagnoses have been made in six cases before 1889, and in as many more since (Wölffler, Bull, Lardy, Tremaine, Karewsky [two cases], Riegner, Annandale, Senn, Küster, Filipow, and the present case).

In the first six cases an accurate diagnosis was made without an exploratory puncture.

In other instances the following diagnoses were made :

Thiersch, *Abscess of the Abdominal Wall.*

Gussenbauer, *Retro-peritoneal Cyst.*

Steele, *Cyst of the Peritoneum.*

Hahn and Kulenkampf, *Hydatid Cyst of Liver.*

Bozeman, Riedel, Koatz, Salzer, Zukowsky, and Martin, *Ovarian Cyst.*

Treves, *Sarcoma.*

In Kulenkampf's case exploratory puncture had withdrawn "a litre of fluid, as clear as limpid water. It contained no echinococci, but was rich in albumen, very alkaline, but free from succinic acid, which may be present in hydatid fluid."

In Treves' case the growth had been rapid. It felt elastic, but did not fluctuate. The idea of a cyst was never entertained; the mass appeared to be solid and heavy.

The essential symptoms to be considered in diagnosing a pancreatic cyst are the presence of a smooth, globular, retro-peritoneal tumour, moving slightly with respiration, in the upper part of the abdomen towards the left side, lying behind the stomach and the transverse colon, and below the former.

The relation of the tumour to the stomach and to the transverse colon may be made out more definitely by distending them with gas, or the colon with water. We would lay especial stress on the great value of this method in the diagnosis of these cases. By the recognition of the stomach in front of the tumour we are able to exclude tumours in connection with the liver, the spleen, and those within the main peritoneal cavity. No renal tumours, unless of enormous size, lie behind the stomach, and these tumours are related to a lateral, and not to a transverse coil of the colon. The diagnosis of ovarian tumour is the most common error; this has arisen rather from the frequency of ovarian cysts than from the difficulty of differentiation. In future, if the relation of the stomach to the tumour is borne in mind, the mistake should be avoided. Some mesenteric cysts, some retro-peritoneal, and occasionally other tumours may present some of the physical signs of pancreatic cysts. In rare cases, therefore, it may be impossible to arrive at a certain diagnosis from the data afforded solely by the situation of the tumour. In any case of doubt the question can be decided, if it is

thought necessary, by means of aspiration, as the fluid which is drawn off is pathognomonic. In the four cases in which incorrect conclusions were formed the examination of the fluid appears to have been incomplete.

As the tension of the tumour increases, pain more or less constant, with associated paroxysmal attacks, becomes a marked symptom; frequently the patient becomes extremely emaciated, and in some instances jaundiced. There is often a history of traumatism.

The fluid which is obtained by aspiration is of a sp. gr. between 1010 and 1020, alkaline, containing 1·5 to 3 per cent. of albumen (with the exception of one case, where the amount was only ·3 per cent.). It is usually turbid, greenish or brownish in colour, but occasionally clear or opalescent and white. It usually, if not always, contains mucin and a sugar ferment; and in some cases tyrosin, blood-pigment, and a trace of urea. It may emulsify fats, and often contains but a small amount of cell-products.

3. *The Liability of Hæmorrhage to take place into Pancreatic Cysts, causing very Grave and Urgent Symptoms, as in our Case.*

In this case the very urgent symptoms, which for a few hours on the night of August 23rd, thirty hours after exploration by abdominal section, so gravely threatened life, were clearly due to hæmorrhage into the cyst, the bleeding being probably due first and foremost to the very vascular surroundings which must always be present with pancreatic cysts; secondly, to the great tension which was so marked a feature in this case; and thirdly, to the inflammatory softening which no doubt was set up by the first operation.

This liability of pancreatic cysts to hæmorrhage is of importance not only in rendering the completion of the operation urgently needed, as in our case, but in explaining certain obscure causes of death. The following cases illustrate the above points. The history of injury in the first and of straining in the second is of interest.

Anger ('Bull. de la Soc. Anat. de Paris,' 1865, 2me série, t. x, p. 192) reports a case of hæmorrhage into a cyst of the pancreas in a man, æt. 72, who had some years before fractured several ribs on the left side, from which injury he recovered completely after three months' treatment in a hospital. Five months previous to admission the lower limbs became œdematous, and for the last six weeks ascites has been present.

On admission into the Beaujon Hospital the following conditions were noticed :—Œdema of the lower extremities, ascites, difficult and stertorous breathing, râles over the left side of the chest, with effusion on the opposite side. The diaphragm was pushed up, the pulse was 100, irregular, and intermittent. Diarrhœa, loss of appetite, and albuminous urine were noted, but no delirium. Two days after his admission the patient died, the pulse having been for a time extremely intermittent and feeble.

Autopsy.—Pleuritic effusion on the right side, bronchitis, the liver somewhat contracted but not cirrhotic. A tumour the size of a fœtal head was found in front of, and on the same level as, the left kidney. The tumour was bounded in front by the stomach and the transverse colon, above by the diaphragm, below by the descending colon, behind by the kidney, towards the middle by the pancreas, and on the outside and above by the spleen. It was loosely joined to the kidney and spleen by connective tissue, in which the vessels of those organs could be distinctly seen. The vessels of the spleen were intimately connected with the posterior surface, and were not easily isolated. The tumour was evidently connected with the pancreas. The external surface was irregular and nodulated, the anterior wall of variable thickness. Fluctuation in the tumour was distinct. On careful inspection the lobules of the pancreas could be separated from the tumour, but the walls of the cyst contained a tissue which resembled glandular structure (*vide infra*). Upon opening the cyst, a considerable quantity of dark fluid blood escaped, which contained a number of unadherent coagula ; the inner surface of the cyst was uneven and reticulated, *i. e.* resem-

bling in appearance the interior of the right ventricle. Diverticula could be seen, which were in free communication with the principal cyst. Microscopical sections of the cyst wall showed cells resembling the pancreas; acinous groups of glandular tissue were also found. M. Anger came to the conclusion that the tumour was a cyst in the tail of the pancreas. The presence of the blood was explained by the supposition that during the progressive dilatation of the cyst some of the vessels in the connective-tissue reticulum had given way. What symptoms the tumour had produced could not be ascertained, as the patient was being treated for heart disease, and the tumour was not recognised during life.

The following case was observed by Störck ('Arch. Gén. de Méd.,' May and July, 1836). The patient was attacked during a menstrual period with vomiting, which was followed by coldness of the extremities, palpitation of the heart, and dyspnœa. Soon after, a pulsating tumour, causing considerable pain, was noticed in the epigastric region. The patient also suffered from attacks of vomiting. She died three and a half months after the first attack. At the autopsy the entire pancreas was found enormously dilated, and weighed with its contents thirteen and a half pounds. It was found filled with coagulated blood, the hæmorrhage having apparently occurred at intervals. The weight of the tumour had caused injurious pressure on other organs. M. Le Dentu believed that the hæmorrhage was caused by the act of vomiting, and had taken place into a pre-existing cyst of the pancreas.

Dr. Parsons ('Brit. Med. Journal,' June, 1857) reports a case where hæmorrhage into a pre-existing cyst proved fatal after the latter had ruptured into the intestinal tract. The patient, æt. 60, had suffered from vague dyspeptic symptoms for an indefinite length of time. A fluctuating tumour, the size of an orange, could be felt in the epigastrium, just below the greater curvature of the stomach. Emaciation progressed rapidly, one day the tumour disappeared suddenly, and at the same time a viscid, dirty-

white fluid was discharged by the bowels. The tumour reappeared in a short time, and ruptured a second time into the intestines, followed by hæmorrhage into the ruptured cyst, which proved fatal. At the *autopsy* the pancreas was found excavated into a wide canal, which at either extremity was converted into a cyst. The walls of the cyst were of the firmness of cartilage, and the organ was adherent to the stomach, kidney, and colon; coagulated blood was found in the dilated duct and cysts.

The following explanations of these cases are suggested by Dr. Senn:

1. Hæmorrhage into a pre-existing pancreatic cyst.
2. Parenchymatous hæmorrhage producing a cyst, followed by hæmorrhage from the cyst wall.
3. The hæmorrhagic cyst may originate in a dilatation of one of the vessels of the pancreas.

4. *Question of the Safety of the Preliminary Puncture from the Front.*

While this step is often the only one, short of an exploratory incision, which will clear up the obscurity of these cases, it is evidently attended with risk. Thus in our case the stomach was certainly traversed by the aspirator needle once, if not twice. The same thing, with like harmlessness, happened in Karewsky's case.

In that case, as in ours, when the abdomen was opened the stomach was found tensely flattened out over the cyst. Not being able to raise the stomach and thus get at the cyst, Karewsky exposed the latter by turning down a flap, making a second incision perpendicular to the first, which was a curved one lying a finger's breadth below the left costal margin.

In some cases, such as Lardy's and Küster's, the preliminary puncture has been followed by evidence of peritonitis; in others (Tremaine) grave collapse has immediately followed. In Cathcart's case rupture of the

cyst, followed by grave collapse, speedily ensued after puncture with a hypodermic needle and withdrawal of about half a drachm of a transparent reddish fluid. Almost immediately after the needle was withdrawn, the patient screamed and writhed with pain. This at first was localised to the point of puncture, but soon spread all over the abdomen. In about half an hour there was flattening where the tumour had existed, with slight bulging in the flanks. The temperature rapidly fell from normal to 97·2°. In about an hour, vomiting of greenish matter came on, and a state of great collapse supervened. Mr. Cathcart determined to open the abdomen at once, but it was not until five hours after the puncture that consent could be obtained. About 40 oz. of dark-coloured, bloody fluid exuded when the peritoneum was opened. By working between the transverse colon and the stomach, the lesser sac of the peritoneum was opened, and a cyst was discovered lying partially collapsed behind the stomach. The cyst was freely opened, when more fluid escaped. As the collapsed cyst could not be easily drawn into the abdominal wound, and as the original opening in the cyst was not to be found, Mr. Cathcart decided to drain it from behind the peritoneum. With this object he explored the cyst's cavity with his finger, and found that below and behind the point of the twelfth rib on the left side, the finger inside the cyst could be pressed directly out against the skin. At this point, therefore, a counter opening was made, and a large drainage-tube passed into the cyst from behind. The cyst and the general peritoneal cavity were then washed out with warm boracic acid solution. A good recovery followed.

Yet another possible risk must be remembered when these cysts are punctured from the front—the presence of the transverse colon. Thus in Lardy's case this bowel was stretched tightly over the cyst. In Rokitansky's case, during an unsuccessful attempt to extirpate the cyst, the transverse colon, which was adherent in front, was torn through. In a case of Billroth's reported by Salzer (l. c.)

the transverse colon masked very completely the lower part of the cyst.

The above interesting case of Mr. Cathcart's leads up to the last point:

5. *The Best Mode of exploring and opening these Cysts.*

While the tenseness with which these cysts bulge against the anterior abdominal wall invites attack from this aspect, and while the great success which has followed treatment of these cysts after opening the peritoneal cavity here (whether by one or two stages) fully justifies a continued use of this method, we think that after Mr. Cathcart's case, and the risks which we have pointed out as following from puncturing in front, an examination should always be made of the infra-costal region behind. If fluctuation is present in this region, or if a thrill can be obtained from the front, the needle should be introduced here, and if the result be successful the cyst should be cut down upon and drained also from this side.

Eleven cases, Nos. 1—11, of pancreatic cyst which had been operated upon are collected in the papers by Senn in the 'International Journal of the Medical Sciences' for 1885 and 1887. It may be convenient to summarise the cases Nos. 12—17, which have been reported in foreign journals.

1. Senn.—Internat. Journal of the Med. Sciences, vol. lxxxix, p. 17.

2. Kulenkampf.—Berliner klin. Woch., February 13th, 1882.

3. Gussenbauer.—Arch. für klin. Chir., vol. xxix, p. 333.

4. Bozeman.—New York Medical Record, January 14th, 1872.

5. Rokitansky.—Wien. med. Presse, November 15th, 1881.

6. Salzer.—Zeit. für Heilk., Prag, vol. vii, p. 11, 1886.

7. Riedel.—Arch. für klin. Chir., Berlin, vol. xxxii, p. 994, 1885.

8. Kramer and Hahn.—Zeit. für Heilk., Prag, vol. vii, p. 25, 1886.

9. Dixon.—New York Medical Record, March 13th, 1884.

10. Luecke.—Virch. Arch., vol. xli, p. 9.

11. Thiersch.—Berlin. klin. Woch., No. 40, 1881.

12. Zukowski.—Wien. med. Presse, No. 45, 1881.

13. Lardy.—Corr. Blatt für Schweitzer Aertze, vol. xviii, p. 279, 1888.

14. Bull.—New York Med. Journal, vol. xlvi, p. 376.

15. Subotic.—All. Wien. med. Zeit., vol. xxxii, p. 279, 1887.

16. Küster.—Deutsche med. Woch., Leipzig, vol. xiii, p. 189, 1887.

17. Wölffler.—Zeit. für Heilk., Prag, vol. ix, p. 119, 1888.

18. Annandale.—Brit. Med. Journ., vol. i, p. 1291, 1889.

19. Cathcart.—Edinburgh Medical Journal, July, 1890.

20. Treves.—Lancet, vol. ii, p. 655, 1890.

Case 12 (Zukowski).—A woman, æt. 36, had suffered with cardialgia and an epigastric tumour for two years and three quarters. A diagnosis of an ovarian tumour was made by Spencer Wells and by Rokitansky.

A partial extirpation was attempted, but the patient died with peritonitis ten days later.

Case 13 (Lardy).—A man, æt. 37, had a year previously been seized with a sudden attack of abdominal pain, which lasted for some hours. After a second attack he noticed a fulness in the epigastrium. He wasted greatly.

In November, 1887, under chloroform, a smooth cyst was punctured above the umbilicus, and a dark red bloody fluid, containing large fatty cells, cholesterin, and red blood-cells, was evacuated. When the stomach and colon were distended with gas, they were shown to be in front of the tumour. The diagnosis of a pancreatic cyst was made. The abdomen was opened and the cyst attached to the abdominal wall and drained, ten litres of fluid being evacuated. The patient recovered.

Case 14 (Bull).—A man, æt. 45, had ten months previously an attack of colic with jaundice. Four weeks later a tumour appeared above the umbilicus, and for ten weeks increased in size. After an attack of abdominal pain and diarrhœa the swelling disappeared; three weeks later it reappeared.

The abdomen, except at the epigastrium and in the right iliac fossa, was dull.

When the stomach was distended with a seidlitz powder its resonant area lay in front of the tumour. A fluid thrill could be obtained across the tumour. The fluid was aspirated with a syringe, and shortly afterwards abdominal section was performed. The cyst wall, which was from half to three eighths of an inch thick, was sewn to the abdominal wall, and opened with a cautery seven days later; 118 oz. of fluid were evacuated. He gained 13 lbs. in a few weeks, but a fistula remained. He died from diabetes two months later. After the tapping a diagnosis of a pancreatic cyst was made. The fluid was of sp. gr. 1010, viscid, and dichroic; being dark greenish brown by reflected, and a dull reddish brown by transmitted light. The deposit was a dark greenish brown. It contained 2½ per cent. of serum-albumen, 2·7 per cent. of glucose, and some mucin. It was diastatic and peptic, and crystals of ammonio-magnesian phosphate were present. It emulsified oil. It was free from urea, peptones, glycogen, bile-pigment, and fat.

Case 15 (Subotic).—A man æt. 20, whose occupation was an arduous one, was seized with attacks of colic and vomiting. An abdominal tumour had been noticed two years previous to his admission, and for one year he had had frequent attacks of vomiting. Œdema of his eyelids and ankles had been present for six months.

On admission the patient was anæmic and emaciated. There was a tumour the size of a child's head, which extended two fingers' breadth below the umbilicus. The upper margin was sharply defined; the surface was smooth,

and covered by the stomach in front, while towards the left the resonance was impaired. Fluctuation could be obtained; the tumour was fixed, did not move with respiration, and transmitted aortic pulsation. The urine contained albumen; the diagnosis was a pancreatic cyst.

March 21st.—The abdomen was opened, and the cyst was attached to the abdominal wall; four days later the cyst wall, which was half an inch thick, was divided with a thermocautery, and two litres of a turbid, brownish, alkaline fluid were let out. It contained fatty nucleated cells, and changed blood-cells. Sugar was present. The albuminuria persisted for some time. The patient completely recovered.

Case 16 (Küster).—A man, æt. 46, had been thrown from a waggon eight years previously and severely shaken. For four months an abdominal tumour had been observed. The cyst was drained, and the evacuated fluid was of a light yellow colour, sp. gr. 1016, and contained 3 per cent. of albumen.

Case 17 (Wölffler).—A girl, æt. 21, with acute gastritis, gave a history of jaundice two years previously, followed by an abdominal tumour. There was no history of injury. She was very feeble, and the tumour occupied both hypochondria; there was tympanitic resonance above due to the stomach, below due to the colon, and between the tumour and the liver due to the duodenum. There was no fat or undigested muscle in the fæces. A pancreatic cyst was diagnosed.

The abdomen was opened and the cyst was drained, the fluid spurting out two feet when the cyst was tapped. Twenty-four hours later the pulse was 140; there was general cyanosis, but no peritonitis. On the fifth day the cyst wall sloughed; on the fourteenth a gangrenous mass of cyst wall the size of a hand was removed. The patient recovered in nine weeks.

The fluid from the cyst was brown, turbid, sp. gr. 1023, and amounted to 2700 c.c. It contained seralbumen, methæmoglobin, a trace of peptone, a sugar ferment, and 1·5 per cent. of albumen. It was free from mucin, metalbumen, bile-pigment, fatty acids, acetone, fat, leucin, and tyrosin. Cholesterin, chlorides, sulphates, and a trace of phosphates were present.

Microscopically there were white and red blood-cells, elliptical endothelial cells with fat drops, and abundant cholesterin crystals.

Appendix (*July*, 1891).

To the above cases may be added the following:

21. Koatz.—Operation einer Pancreascyst, Dissertation, Marburg, 1886.

22. Fenger.—Chicago Medical Journal, February, 1888.

23. Steele.—Chicago Medical Journal, April, 1888.

24. Tremaine.—Transactions of the American Surgeons' Association, vol. vi, p. 557, 1888.

25. Ochsner.—Arch. für clin. Chir., vol. xxxix, p. 442.

26 and 27. Karewsky.—Deutsche med. Woch., No. 46, 1890.

28. Filipow.—St. Petersburg Med. Woch., No. 9, 1890.

29. Riegner.—Berliner klin. Woch., No. 42, 1890.

30. Martin.—Virch. Arch., vol. cxx, 1890.

Case 21 (Koatz).—A woman, æt. 36, developed a large abdominal cyst, which on puncture yielded a brown liquid, sp. gr. 1020. The abdomen was opened in 1885, but extirpation appearing impossible, nothing further was done. In 1886 a partial extirpation was attempted; the patient died seven weeks later with pericarditis and pyelo-nephritis.

Case 22 (Fenger).—A boy, æt. 8, fell from a horse and bruised his abdomen. For eight weeks he had intermittent

abdominal pain; three weeks after his accident a tumour, which has slowly increased in size, appeared in the epigastric region. Aspiration evacuated a turbid, alkaline, yellowish fluid.

No diagnosis was made. The abdomen was opened, and a pancreatic cyst contained 40 oz. was drained the following week. At the end of three weeks there was only a small fistula.

Case 23 (Steele).—William J—, æt. 40. At the age of twenty-seven he was crushed between two railway waggons; in the following year a cyst gradually developed in the umbilical region. This was aspirated, and 52 oz. of chocolate-coloured fluid was drawn off. This aspiration was repeated four times during the succeeding ten years.

The patient wasted. The tumour was dull on percussion. He had attacks of colicky pain.

The abdomen was opened, and the cyst was attached to the anterior abdominal wall, and was opened with the cautery later. The wall was three quarters of an inch thick. The fluid evacuated contained albumen, fatty matter, and oxalate of lime, but it could not be shown to possess any digestive properties. After three months the patient's condition was good, but the sac did not close up. It was proposed to drain it further through the loin.

At the time of the operation, examination with a finger showed that the cyst occupied the tail of the pancreas.

Case 24 (Tremaine).—A man, æt. 30, developed an epigastric cyst, which was punctured. A fluid having the properties of pancreatic fluid was removed; it had a sp. gr. 1007, with 10 per cent. of albumen, ½ per cent. of sodium chloride, and a trace of sugar. Collapse, due to rupture of the cyst, supervened, the abdomen was opened, the cyst was sutured to the abdominal wall, and the wound closed. The patient recovered; the wound healed, but soon the cyst refilled. The lower extremity of the cyst was then drained, and the patient was cured in three months.

Case 25 (Ochsner).—A patient, æt. 24, had for four years noticed a cyst to the left of the umbilicus. Six months after a pregnancy it rapidly increased to the size of a full-term uterus. The cyst was smooth, moveable, and tense, occupying the left hypochondriac region; it was dull on percussion, but at times lay behind intestinal coils. No diagnosis was made.

The abdomen was opened. The omentum and some coils of intestine which presented were pushed up; fourteen litres of fluid were evacuated, and the cyst was attached to the abdominal wall. The drainage-tube was removed at the end of three months, and the wound was closed a month later. At the time of the operation, veins the size of a finger were visible on the cyst wall.

Case 26 (Karewsky).—A man, æt. 25, injured the left side of his abdomen through a fall, in consequence of which he was confined to his bed with attacks of vomiting.

Four weeks after this an abdominal tumour appeared, and gradually increased in size. On percussion it was dull; it was bounded above and in front by the stomach, below by the colon. A trocar was passed through both walls of the stomach, and 50 c.c. of a brown, alkaline, albuminous fluid was drawn off; it did not possess any diastatic properties.

In eight days the cyst had increased in size, the abdomen was opened, the cyst was incised, and the walls were sutured to the abdomen. Recovery in five weeks.

The sp. gr. of the liquid, which was only faintly alkaline, was 1012; it contained albumen, globulin, and propeptone. It was amylolytic, and digested fibrin.

The fluid from the fistula was strongly alkaline, clear as water; it was amylolytic, but did not digest fibrin; it emulsified fats.

Case 27 (Karewsky).—A man æt. 58, in consequence of an injury to his left costal margin, developed vomiting and epigastric pain. After three months a tumour appeared

to the left of the epigastrium. Two months later, when examined, the tumour was made out to be a cyst behind the stomach and colon, and was diagnosed as a pancreatic cyst.

The abdomen was opened, but the extreme tension of the cyst rendered it impossible to attach it to the abdominal wall. It was therefore incised and 3 litres of fluid evacuated, part of it escaping into the abdominal cavity.

The cyst rapidly disappeared, but a fistula remained, from which there daily discharged about 500 c.c. of pancreatic fluid, which digested the edges of the wound. This discharge suddenly ceased, and the patient soon recovered. The fluid from the cyst neither peptonised proteid nor emulsified fats, but was strongly amylolytic. The fluid from the fistula possessed all the properties of pancreatic fluid.

Case 28 (Filipow).—A woman, æt. 65, had noticed for three years an epigastric tumour, which for six months had increased in size, and had caused vomiting, wasting, and paroxysmal pains with slight jaundice. The tumour was a cyst, dull on percussion, situate behind the stomach and separate from the liver and spleen. The diagnosis lay between a pancreatic and a mesenteric cyst, probably the former.

The abdomen was opened; the cyst was partially resected, and sutured to the abdominal wall. The fluid was alkaline and dark brown; the cyst was found to be connected with the head and part of the body of the pancreas. The patient left eighteen days after the operation with a small fistula.

Case 29 (Riegner).—A girl, æt. 23, fell from a great height, after which she suffered with attacks of abdominal pain. Three years later the pain became more severe, she became slightly jaundiced, and had severe attacks of vomiting. An epigastric swelling which steadily increased in size was observed; this was formed by a dilated stomach over a cyst. The fluid removed by aspiration was alkaline,

brown, emulsifying fats, amylolytic, and containing red and white corpuscles. A pancreatic cyst was diagnosed.

The abdomen was opened; on puncturing the cyst the fluid rushed out with such violence as to inundate the table; 1500 c.c. of fluid were removed. The patient completely recovered.

The fluid removed was of sp. gr. 1009, alkaline, emulsified fats, and was amylolytic, but did not digest proteids. The fluid which drained from the fistula after the operation digested fibrin.

Case 30 (Martin).—In 1873 Dr. Martin's father attempted to remove what was supposed to be an ovarian cyst from a woman æt. 33, but, in consequence of the number of adhesions, the abdominal wound was merely closed. A small tumour to the left of the umbilicus had been noticed after her first confinement twelve years previously. In 1890 the abdomen was enormous, measuring sixty-two inches in circumference. A cyst filled the abdomen, and lay behind the stomach and colon; the patient had not wasted. The abdomen was opened by Dr. Martin, and the cyst detached from the adherent viscera; it was incised, and 15 litres of fluid evacuated. The pedicle, which was very vascular, was ligatured and divided. The patient left the hospital in twenty-seven days. The cyst was connected with the pancreas; the walls were nearly 3 mm. thick; it contained innumerable secondary cysts, lined by cylindrical epithelium. The fluid contained albumen and cholesterin, and, in some cysts, blood.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 169.)

THE CAUSATION AND MODE OF PRODUCTION OF PES CAVUS.

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THE deformity known as pes cavus, hollow foot, or clawed foot, is one that apparently has not attracted very much attention, and the causation of which has not been indicated very satisfactorily. In fact, not a little confusion exists about its mode of production, and the statements given in books are contradictory and misleading; in many recognised text-books it is not described at all, in others it is disposed of in a few words; and yet cases are by no means uncommon, especially in association with other forms of talipes.

The deformity consists in an increased height of the longitudinal plantar arches, with a corresponding increase in the convexity of the dorsum, the scaphoid and cuboid bones being often very prominent, and occasionally covered by a bursa. The ball of the great toe is more conspicuous than usual, and it, along with the under surfaces of the other toes, is approximated to the heel, so that an antero-posterior diminution of the length of

the foot, varying from a half to one and a half inches, is produced. The central portion of the plantar fascia is thickened and contracted.

Accompanying this condition is a subluxation of the first phalanges of the toes upon the heads of the metatarsals, in severe cases the phalanges being almost vertical; the terminal phalanges are flexed. The tendons of the ext. proprius hallucis, ext. longus digitorum, and tibialis anticus often stand out conspicuously, and an excessive movement of the toes occurs when attempts are made to extend the foot. This condition is almost invariably associated with talipes equinus, and the tibial articular surface of the astragalus can often be felt projecting on the dorsum. Corns develop on the under surfaces of the heads of the metatarsal bones, and sometimes over the first interphalangeal joints.

The explanations given in various books of the origin of this condition may be briefly considered.

The writer in Heath's 'Dictionary of Surgery,' speaking of talipes equinus being due to contraction of the tendo Achillis, says, "In the majority of cases nothing else is at fault, but in some, a further examination being made, the arch of the foot is found unduly flexed and the plantar fascia to be tightly contracted."

In 'Holmes' Surgery' the writer says, "As a substantive deformity I am not acquainted with it [pes cavus]."

Erichsen quotes Duchenne as saying, "When the interosseous muscles are paralysed or atrophied, the tonic contraction of the muscles which extend the first phalanges and that of the muscles which flex the last phalanges being no longer opposed, the claw-like condition of the toes becomes augmented. The posterior extremities of the first phalanges are subluxated upon the heads of the metatarsal bones; then the curve of the plantar arch becomes increased and the plantar fascia shortened," &c.

Mr. Golding-Bird, in the 'Guy's Hospital Reports' for 1883, ascribes the deformity to weakness or paralysis of the peronei muscles. In 'Holmes' System' and in

'Gant's Surgery' I can find no mention of the condition.

That Duchenne is wrong in ascribing the deformity to paralysis of the interossei seems at once evident from the tilting of the toes being only a small part of the affection, and personally I am quite unable to follow out his explanation of how this increases the height of the plantar arch. The extensor proprius hallucis and longus digitorum can only extend the foot when the toes are extended, or when they are fixed by the flexors; and the actions of these muscles must be weakened by the approximation of their attachments.

Besides, this extension of the foot tends to flatten the arch rather than to deepen it. Then, again, no cause for paralysis of the interossei is suggested, and infantile paralysis of these muscles as an isolated condition is scarcely conceivable. A still stronger argument is that interosseal paralysis of the hand does not produce a similar deformity, although the arrangement of muscles is the same.

Mr. Golding-Bird's suggestion that the peronei muscles are at fault would account, perhaps, for the production of talipes varus from over-action of the tibial muscles; but that the over-action of these muscles should be sufficiently great to produce an actual bony deformity of the tarsus seems improbable. Spasm of muscles may cause a temporary deformity, but a permanent change is surely more likely to be caused by fibroid contraction of a paralysed muscle than by spasm of an opponent.

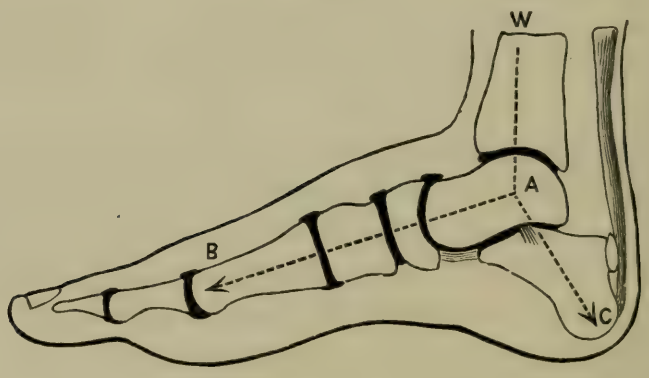
The affection under consideration has been also ascribed to wearing tight boots. That this should produce a diminution in the length of the foot of from a half to one and a half inches is inconceivable, especially as the condition is often unilateral. Another explanation offered is that of the combined over-action of the tendo Achillis and ext. longus digitorum. If this were true we should expect to find the deformity most marked in those who have not walked, as there is always a tendency in walking to

flatten out the arches of the foot; it will be shown later that this is not the case. That a contraction of the plantar fascia could *per se* cause the deformity is very unlikely; if it were so we should expect to find the inner portion of the fascia the most thickened. Besides, granting this, what is the cause of the fascial contraction?

These explanations, which I have taken *seriatim*, are obviously very unsatisfactory, and it is necessary to see whether a better one cannot be offered. In order to do this it will be advisable to consider how the weight of the body in the erect position is supported by the plantar arches.

Referring to Fig. 1, the weight of the body, passing vertically to a point A, is then transmitted to the ground

FIG. 1.

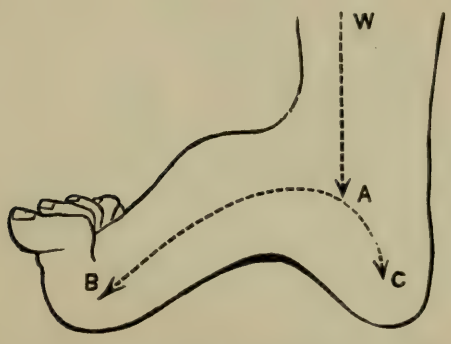


at the points of contact, viz. the heel and the balls of the toes. The weight of the body W, acting along W A, may thus be represented as broken up into two components, A C and A B, of which A C is the greater. But each of these may be again broken up into a vertical and a horizontal part; and whilst the vertical components represent the proportion of W which belongs to each, the horizontal components tend together to diminish the height of the arch, as they act in opposite directions.

If now we apply the same process to a case of talipes equinus, Fig. 2, we find that owing to the tilting of the

heel, which cannot be made to touch the ground, the weight of the body is transmitted along A B only, and A C represents a latent force. This force cannot be neutralised by any pull of the tendo Achillis, for this would simply transfer again the vertical portion of A C to the tibia and femur. The action of the contracted soleus and gastrocnemius is simply to limit the movement at the ankle-joint in the direction of extension of the foot. If now A be taken as situated in the centre of the astragalus there is a constant tendency for that point to move nearer C; and the point B being fixed by contact with the ground, and therefore the horizontal component of A B neutralised,

FIG. 2.



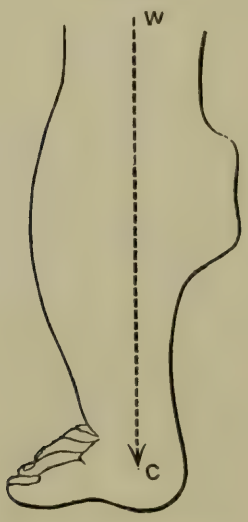
the force A C is constantly tending to bend or render convex upwards the portion of the arch A B. This tendency is resisted by the dorsal and interosseous ligaments of the foot and the shape of the bones of the metatarsus and tarsus. Any muscular resistance must of necessity be small and easily compensated for by muscular or tendinous adaptation.

The force A C tending to produce convexity of the tarsus is not, however, constant; in a position of rest it does not exist, for a patient with talipes equinus will flex the thigh and leg so that the centre of gravity may pass through the point B, *i. e.* through the heads of the metatarsal bones. It is only in walking that the mechanism described comes into play, and it follows from this that a person with talipes equinus would only develop pes cavus after he had been

walking for some time ; and that a patient who does not walk or who is confined to bed would not develop pes cavus at all, but would only have the talipes equinus increased by the weight of the foot. Similarly a patient with talipes equinus who wears a high-heeled boot will not develop pes cavus, the force A C being directly transmitted to the ground.

Considering the mechanical forces further, it will be readily seen that the greater the talipes equinus the less the tendency to develop pes cavus ; so that if the heads of the metatarsals fall directly in the line of the transmitted body-weight, the tendency to formation of pes cavus is nil. This, represented in Fig. 3, is found to be the case

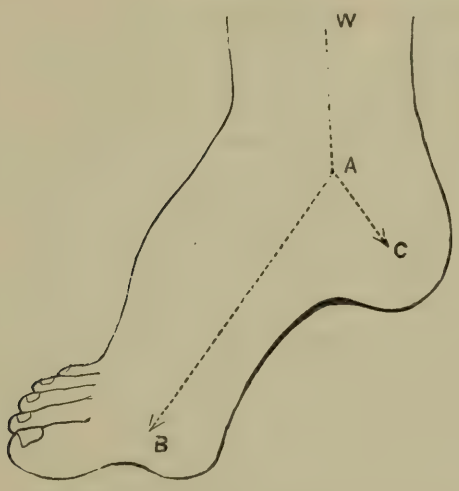
FIG. 3.



in practice, and I have been unable to find any case like Fig. 3 in which there was mention of contracted plantar fascia, &c. Again, the force tending to increase the curve of the plantar arch being greatest as soon as the heel is raised slightly from the ground, it follows that the bending of the arch is really a compensatory change allowing the heel to again touch the ground. When this happens equilibrium is re-established, only to be again rendered unstable by a further contraction of the soleus

and gastrocnemius. This condition of established equilibrium is represented in Fig. 4; and it may be here noticed that, in those cases of pes cavus without any apparent equinus, not only is the tendo Achillis tense, but extension of the foot beyond a right angle is impossible. In the majority of cases, however, compensation cannot occur to a sufficient extent, and along with the pes cavus

FIG. 4.



there is still talipes equinus, which may or may not be associated with varus, and probably in rare cases with talipes valgus, though of the latter I am a little uncertain.

The cause of the talipes equinus varies in different cases; in some it is congenital, in the great majority associated with some talipes varus. In acquired cases infantile paralysis of the soleus and gastrocnemius, followed by fibroid contraction of these muscles, is a common cause. Other causes of talipes equinus need not be enumerated, but it is not too strong a statement to make that any case of talipes equinus, however produced, may be associated with pes cavus, provided only that the patient transmits the weight of the body for a sufficient length of time through the affected foot without the intervention of a raised artificial heel.

The characteristic condition of the toes has from time to time been noticed from its resemblance to "hammer-toe," and hence probably the supposition that pes cavus was due to wearing too short boots.

Primarily the first phalanges are hyperextended—or rather one might say that the metatarsals are hyperextended on the phalanges—when the foot acquires the position of talipes equinus, the condition represented in Fig. 2. Consequently the extensors of all the toes shorten to adapt themselves to the new position. As soon as this hyperextension from position has reached a certain stage (and not only is there hyperextension, but an actual subluxation upwards with expansion of the articular surfaces of the metatarsals) the interossei and lumbricales are rendered inactive, or act in a direction the reverse of normal, because their lines of action are now behind the centre of the heads of the metatarsals. When these muscles act they tend actually to increase the deformity, further extending the first phalanges and not extending the terminal phalanges, which become flexed either by their own weight, by adaptation to the boots, or by the pull of the flexor tendons. This mechanism is obvious on considering the anatomy of the part, and is exactly similar to what occurs in hammer-toe ; in fact, Mr. Lane has pointed out in the 'Trans. Path. Soc.,' 1886 ("Causation and Pathology of Rheumatoid Arthritis"), that even the flexor tendons may become displaced into the grooves between the heads of the metatarsals instead of passing over them.

The interossei being rendered useless in this way probably waste to a certain extent, accounting for their supposed paralysis, which is really an effect of the changes in the foot, and not the cause of them.

The characteristic tilting of the toes, and especially of the big toe when attempts are made to extend the foot, is due to the action of the extensors, no longer opposed, but rather aided by the interossei, while extension of the foot is resisted by the tendo Achillis.

Though the extensors are shortened to a certain extent,

there is no evidence that they have any primary action in the production of the deformity under consideration ; their range of movement is good, though the effect produced is small.

It is obvious, on comparing Figs. 2 and 4, that the condition of the toes will be more conspicuous in talipes equinus with pes cavus than in talipes equinus alone ; the extreme condition is seen in Fig. 3. To sum up, the deformity of the toes is due primarily to the foot being in the equine position, and secondarily to the displacement of the interossei and contraction of the extensor muscles.

The weight of the body being transmitted through the heads of the metatarsals causes, as a rule, considerable discomfort, but in several cases I have seen the patients did not suffer much pain until the curving of the plantar arch, stretching of ligaments, alteration in shape of bones, &c., were taking place ; then with these were associated corns beneath the heads of metatarsals and over the first phalangeal joints, with bursæ on the dorsum.

It follows from what has been said that there is an approximation of the bases of the toes to the heel, consequently the plantar fascia contracts to accommodate itself, this being again a result, not a cause of the deformity.

The contraction of the tibial muscles, anterior and posterior, that is sometimes seen is either a shortening due to approximation of attachments, or is a true shortening, the result of fibroid changes after infantile paralysis ; in the latter case there will of necessity be a more or less marked varus added to the equinus or equino-cavus. A similar statement will apply to the peronei muscles. That the deformity might be due to a combined overaction of the tibial and peronei muscles is negatived by the occurrence of pes cavus in those only who walk with the affected foot.

My contention is, then, that pes cavus is a secondary deformity, resulting from the transmission of the weight of the body to the ground through a foot in the position of talipes equinus. It is a secondary deformity in that it

is consequent on talipes equinus or equino-varus, and in slight cases it is compensatory. This compensatory arrangement is, however, limited.

The following statistics, taken from the 'Guy's Hospital Reports' for the last five years, well bear out my conclusions:

Of 53 cases of talipes equinus or equino-varus with or without pes cavus, 15 were congenital, 38 were acquired.

A. *Congenital Cases.*

Eleven cases of talipes equino-varus below three years of age; 3 cases of talipes equino-varus with pes cavus, aged seven, twelve, thirteen years; one case of talipes equino-varus without pes cavus, aged seven years.

In the last case, placed separately as an apparent exception, the presence of equinus was extremely doubtful, so that this apparent exception is really confirmatory.

B. *Acquired Cases.*

Four cases of talipes equinus with or without varus below four years of age; 1 case of *very extreme* talipes equinus, no pes cavus, aged seven years; 2 cases of pes cavus, in one of which it is definitely stated that power of extension of foot was absent; 16 cases of talipes equino-varus with pes cavus, the ages of which were—four and a half years, 2; six years, 2; seven years, 1; eight years, 3; twelve years, 2; fifteen years, 2; after fifteen years, 4.

One child, aged seven, with talipes equinus and slight varus, had no pes cavus; he had worn a high-heeled boot for some years.

It may be argued, of course, that these figures give no idea of the date when the deformity began, but the condition of pes cavus being a gradual development, such a date cannot be definitely fixed in any case. What is important is that in no case was pes cavus noticed before four and a half years of age, *i. e.* about three years after

first walking, a point strikingly suggestive of an acquired deformity. The preponderance of cases between the ages of five and fifteen years is obviously due to the bones of the tarsus and metatarsus being more mouldable, and capable of better adaptation to altered conditions, in early than in later life.

In the three congenital cases it appeared as though the primary condition was that of talipes equinus, and the cavus developed as an acquired condition after the patient had learnt to walk. Cases of congenital high arch do occur, but in these there is probably no deformity of the toes ; in fact, there is no reason why such should develop in a primary pes cavus, supposing such a condition to exist.

The conclusions justified by the above figures seem to me to be—1. That pes cavus is a secondary deformity engrafted upon talipes equinus with or without varus.

2. That pes cavus is not congenital, although it may follow upon congenital talipes equinus or equino-varus.

3. That it is the direct result of the transmission of the weight of the body to a foot in the position of talipes equinus, and is the result of a compensatory change, tending to bring the heel to the ground.

4. That the changes in the soft parts, and in the position of the toes, are due to alterations in the arrangements of the tarsal and metatarsal bones, *i. e.* are consequent on the deformity.

NOTE.—In the absence of the author there was no discussion on this paper.

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